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OPHTHALMOLOGICAL TRANSACTIONS

VOL. XXXVII

TRANSACTIONS

OF THE

OPHTHALMOLOGICAL SOCIETY

OF THE

UNITED KINGDOM

VOL. XXXVII

SESSION 1917

WITH

LIST OF OFFICERS, MEMBERS, ETC.

LONDON
J. & A. CHURCHILL
7, GREAT MARLBOROUGH STREET

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NOTICE.

The present volume comprises the proceedings of the Ophthalmological Society of the United Kingdom during its Thirty-seventh Session, May, 1917.

The Society does not hold itself responsible for the statements, reasonings, or opinions expressed in the communications which the Council has deemed suitable for publication.

TABLE OF CONTENTS

OF VOLUME XXXVII.

	PAGE
Officers and Council	IX
List of Presidents	X
List of Members	XI
Rules	XXXIV
List of Communications made during the Session	XLI
List of Plates	XLV
List of Diagrams, etc., in Text	XLVI
Communications	1
Appendix and Museum Specimens	381
Report of Council	383
Balance Sheet	385
Index	387

OFFICERS AND COUNCIL

OF THE

Ophthalmological Society of the United Kingdom

ELECTED AT

THE ANNUAL GENERAL MEETING, MAY 3RD, 1917.

President.

E. TREACHER COLLINS.

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PRESIDENTS OF THE SOCIETY
(FROM ITS FORMATION).

ELECTED	
1880	SIR WILLIAM BOWMAN, Bart., LL.D., F.R.S.
1883	SIR JONATHAN HUTCHINSON, F.R.S.
1886	JOHN WHITAKER HULKE, F.R.S.
1889	JOHN HUGHLINGS JACKSON, M.D., LL.D., F.R.S.
1890	HENRY POWER.
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ELECTED	
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1885	J. HUGHLINGS JACKSON, M.D., LL.D., F.R.S.
1886	PROF. ZEHENDER (Rostock).
1887	HENRY POWER.
1888	SIR HENRY R. SWANZY.
1889	PROF. HANSEN GRUT (Copenhagen).
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1892	PROF. LEBER (Heidelberg).
1893	T. PRIDGIN TEALE, F.R.S.
1895	SIR W. R. GOWERS, M.D., F.R.S.
1896	PROF. SNELLEN (Utrecht).
1898	PRIESTLEY SMITH.
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1904	F. W. MOTT, M.D., F.R.S.
1907	PROF. SATTLER (Leipzig).
1909	EDWARD NETTLESHIP, F.R.S.
1911	E. LANDOLT, M.D. (Paris).
1914	PROF. W. UHTHOFF (Breslau).
1917	SIR GEORGE BERRY, M.B., LL.D.

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1907	JOHN HERBERT PARSONS.
1909	EDWARD NETTLESHIP.
1912	GEORGE COATS.
1915	EDWARD TREACHER COLLINS

*** *Members are requested to communicate with the Medical Secretary when corrections are necessary.*

LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

Professor Leber, Heidelberg.
Professor Fuchs, Vienna.
Professor Sattler, Leipzig.
Thomas Buzzard, M.D., 74, Grosvenor Street, W.
E. Landolt, M.D., Paris.
Professor Gullstrand, Upsala, Sweden.

EXPLANATION OF ABBREVIATIONS.

O.M.—Original Member.
Pres.—President.
V.-P.—Vice-President.
T.—Treasurer.
L.—Librarian.
S.—Secretary.
C.—Member of Council.
*.—Denotes Resident Life Members.
†.—Denotes Non-Resident Life Members.

GENERAL LIST OF MEMBERS.

ELECTED

1905 Adams, P. E. H., M.B., 53, Broad Street, Oxford.
1912 Alexander, G. F., M.B., 35, Prince of Wales Terrace, Scarborough.
1907 Allport, Wilfred, M.B., 133, Edmund Street, Birmingham.
1899 †Amenabar, Daniel, Casiila 699, Santiago, Chili.
1910 Angelucci, Prof., 2, Piazza N. Amore, Naples.
1913 Anklesaria, M. Dhanjishaw, Ahmedabad, India.
1891 †Armstrong, Hugh, Tamworth, New South Wales.
1895 Attlee, John, M.D., 65, Grosvenor Street, W.
1910 Axenfeld, Prof., Freiburg i.B., Germany.

ELECTED

1911 Baker, George Wiston, M.D. (Address uncommunicated.)

1903 Ballantyne, A. J., M.D., 11, Sandyford Place, Glasgow. (C. 1914–17.)

1907 Bardsley, Percy C., M.B., 67, Wimpole St., W.

O.M. *Barlow, Sir Thomas, Bart., K.C.V.O., M.D., 10, Wimpole Street, W. (C. 1880-81. V.-P. 1894-7.)

1889 †Barrett, James W., M.D., 34, Collins Street East, Melbourne, Australia.

1917 Barrie, T. Stewart, M.B., 23, Glanville Street West, Glasgow.

1883 Barton, J. Kingston, 14, Ashburn Place, Courtfield Road, S.W.

1892 Batten, Rayner D., M.D., 9, Wimpole Street, W. (C. 1905-8.)

1888 *Beaumont, W. M., 4, Gay Street, Bath. (V.P. 1912–15. C. 1901-4.)

1912 Beedham, Henry Wm., M.B., 15, Crediton Hill, West Hampstead, N.W.

1909 Bell, T. Herbert. (Address uncommunicated.)

1891 †Bennett, Alfred H., 16, North Terrace, Adelaide, South Australia.

1897 Bennett, H. Percy, M.B., 12, Victoria Square, Newcastle-on-Tyne.

1905 Bennett, F. D., M.D., 18, Saville Row, W.

O.M. Berry, Sir George A., LL.D., M.B., 31, Drumsheugh Gardens, Edinburgh. (*Pres.* 1909-11. C. 1889-92. V.-P. 1895-8. 1912.)

1899 Bickerton, R. E., D.S.O., M.B., 137, Harley Street, W.

1881 *Bickerton, T. H., 88, Rodney Street, Liverpool. (C. 1895-8. V.-P. 1910-12.)

1905 Bird, J. W., D.S.O., 15, Downside Crescent, Hampstead, N.W.

1892 Black, John Wilson, M.D., 46, Academy Street, Inverness.

1898 Blair, Charles Samuel, M.D., 14, Welbeck Street, W.

ELECTED

1917 Bodkin, Herbert, c/o Capital and Counties Bank, Chelmsford.

1895 †Bonar, Thomson, M.D., Elm Lodge, Staplegrove - Taunton, Somerset.

1907 Bond, C. Shaw, M.B., Liphook, Hants.

1911 Boucher, R. B., M.D., Vancouver, B.C.

1885 Bower, Ernest Dykes, M.D., Elton House, Gloucester.

1897 Bower, John A., M.B., 18, Royal Crescent, Cheltenham.

1905 Brailey, A. R., 11, Old Burlington Street, W.

1902 *Brailey, Wm. Herbert, M.D., 21, Lansdowne Place, Sussex.

1911 Brearley, E. A., M.B., University Club, Sydney, N.S.W.

1911 Brennard, H. T. W., University Club, Sydney, N.S.W.

1899 Brewerton, Elmore, 73, Harley Street, W. (C. 1909–10; 1914–17. S. 1910-13.)

1903 Brinton, A. G., Box 5852, Johannesburg, South Africa.

1909 Broachâ, B. B., Karachi Eye Hospital, Karachi, India.

1886 Bronner, Adolph, M.D., 33, Manor Row, Bradford. (C. 1900-3.)

1901 *Brooks, R. Philip, 14, Welbeck Street, W.

1895 Browne, J. Walton, M.D., 10, College Square North, Belfast.

1901 Buchanan, Leslie, 8, Royal Crescent West, Glasgow.

1907 Buckland, Francis, M.D., Moorland Court, Poole Road, Bournemouth.

1907 Burdon-Cooper, J., M.D., 12, The Circus, Bath.

O.M. †Burnham, G. H., M.B., 157, Simcoe Street, Toronto, Canada.

1909 Butler, Thomas H., M.D., B.Ch., 26, Adelaide Road, Leamington.

1902 Buzzard, E. Farquhar, M.D., 78, Wimpole Street, W. (C. 1908-9. S. 1909-12.)

1897 †Byers, W. Gordon M., M.D., 346, Mountain Street, Montreal.

1892 †Caiger, Herbert, M.B., Burghersdorp, Cape Colony.

1905 Campbell, C. A., 93, Bloor Street E., Toronto.

ELECTED

1891 Campbell, E. Kenneth, M.B., 23, Wimpole Street, W.

1891 Cargill, L. Vernon, 35, Cavendish Square, W. (C. 1905-8.)

1912 Carr, Edward, Laround, Colne, Lancs.

1895 Cartwright, E. H., M.D., c/o Union and Smiths Bank, 66, Charing Cross, S.W.

1911 Chambers, W. J., 212, VII Avenue W., Calgary, Alberta, Canada.

1885 Clarke, Ernest, M.D., B.S., 3, Chandos Street, Cavendish Square, W. (C. 1893-6. V.-P. 1910-12).

1911 Clegg, John Gray, M.D., 22, St. John Street, Manchester.

1905 Clements, E. C., 3, Lindum Road, Lincoln.

1907 Cobbledick, Arthur Stanley, M.D., B.S., 21, Bolton Street, Mayfair, W.

1885 Collins, E. Treacher (*Pres.*), 17, Queen Anne Street, W. (V.-P. 1905-8; 1916-17. C. 1889-92; 1901-4. S. 1898-1901.)

1886 Collins, Sir W. J., K.C.V.O., M.S., M.D., 1, Albert Terrace, Gloucester Gate, Regent's Park, N.W.

1901 Cooke, Arthur, M.B., 69, Bridge Street, Cambridge.

1901 Cooper, Ludford, M.D., 19, Victoria Street, Rochester, Kent.

1909 Coorey, Edward A., M.D., Bolton, Bainbalapitiya, Ceylon.

1894 †Coote, Patrick, M.D., 73, St. Ann Street, Quebec, Canada.

1914 Coppez, Henri, M.D., 24 Avenue des Arts, Brussels.

1904 Corbett, W. J., 18, Weymouth Street, W.

1898 Coulter, Robert J., M.B., 15, Stow Park Crescent, Newport, Mon.

O.M. Couper, John, M.D., 80, Grosvenor Street, W. (C. 1881-2. V.-P. 1895-8.)

1895 †Courtenay, J. D., M.D., Ottawa, Canada.

ELECTED

1909 Craig, James A., M.B. (C.), 11, University Square, Belfast.

1901 Crawley, F. C., M.D., 5, Fitzwilliam Place, Dublin.

1907 Cree, Robert Ewart, M.D., 40, Jesmond Road, Newcastle-on-Tyne.

1899 Cresswell, F. P. S., M.B., B.S., 24, Windsor Place, Cardiff.

1904 Cridland, A. Bernard, Salisbury House, Chapel Ash, Wolverhampton.

O.M. Critchett, Sir G. Anderson, Bart., C.V.O., 21, Harley Street, W. (*Pres.* 1899-1901. C. 1883-6. V.-P. 1894-7, 1902-5.)

1898 Critchley, Harry G., M.D., 83, Park Lane, Croydon.

1906 Crocket, A. Pierce. (Address uncommunicated.)

1881 Cross, F. R., M.B., LL.D. (V.P.), Worcester House, Clifton, Bristol. (*Pres.* 1913–15. V.-P. 1898-1901. C. 1891-4.)

1902 Cruise, R. R., C.V.O. (C.), 85, Harley Street, W.

1912 Cummins, J. D., M.B., 10, Clare Street, Dublin.

1904 Cunningham, H. H. B., M.D., 30, University Square, Belfast.

1905 Cunningham, Major J. F. (C.), R.A.M.C., B.E.F., France.

1916 Danis, Marcel, M.D., La Panne, Belgium.

1907 Davidson, Alex. Dyce, M.B., "Hollydene," Cholmeley Park, Highgate, N.

1917 Davidson, Isaac A., M.D., 3, Laurington, Antrim Road, Belfast.

1884 Davidson, Sir James McKenzie, M.B., 26, Park Crescent, W. (C. 1892-5.)

1903 Davies, David, M.B., 8, Lonsdale Gardens, Tunbridge Wells.

1908 Davies, D. L., M.S., M.D., 31, Newport Road, Cardiff.

1889 †Davis, G. C., 173, Liverpool Street, Sydney, New South Wales.

1897 Dawnay, Arch. Hugh Payan, 126, Harley Street, W.

ELECTED

1909 Dé, Chandra Mohan (Rai Bahadur), Med. School, Agra, U.P., India.

1914 De Lapersonne, Prof., M.D., 30 Rue de Lisbonne, Paris.

1903 Dean, C. W., Lindow Square, Lancaster.

1903 Dickson, R. H., Nelson Place, Newcastle-under-Lyne, Staffordshire.

1910 Dighton, Chas. A. Adair, M.B., 73, Rodney Street, Liverpool.

1910 Dimmer, Prof., 15 Reichsratstrasse, Vienna.

1881 Dixon, W. E., Oulton Lodge, Oulton Broad, Lowestoft.

1889 Dodd, Henry Work, 136, Harley Street, W. (C. 1902-5).

1907 Dombrain, Ernest A., 205, Macquarie Street, Sydney, N.S.W.

1905 Dorrell, E. A., 45, Welbeck Street, W.

1899 Douglas, J. Sholto, M.B., Mara Berbice, British Guiana.

1881 *Drake-Brockman, E. F., 18, Hanover Street, W. (C. 1895-8.)

1889 †Drake-Brockman, Herbert E., I.M.S., c/o Thomas Cook and Son, Ludgate Circus, E.C.

1895 Du Boulay, H. H., 2, Royal Terrace, Weymouth.

1909 Duggan, J. N., Sir J. C. Ophthalmic Hospital, Girguina, Bombay.

1886 Dunn, Percy, 54, Wimpole Street, W.

1900 Eason, Col. H. L., M.B. (C.), R.A.M.C., 62, Portland Place, W.

O.M.*Edmunds, Walter, M.B., 2, Devonshire Place, Portland Place, W. (C. 1885-8.)

1902 †Elliot, Robert Henry, Lt.-Col. I.M.S., M.D., Sc.D., 54, Welbeck Street, W.

1903 Ellis, W. F., c/o Messrs. Holt, 3, Whitehall Place, S.W.

1908 Emmerson, Herbert H., 275, Glossop Road, Sheffield.

1883 *Emrys-Jones, A., M.D., 10, St. John Street, Manchester.

1900 Evans, J. Jameson, M.D., 41, Newhall Street, Birmingham.

1902 Eversheb, A. R. F., 22, Harley Street, W.

ELECTED

1907 Fenton, Capt. A., M.B., I.M.S., c/o Messrs. Thos. Cook, and Son, Rangoon.

1911 Fenwick, George, Auckland Club, Auckland, N.Z.

1888 Fergus, A. Freeland, M.B., 22, Blythswood Square, Glasgow. (C. 1905-8.)

O.M. †Ferguson, H. L., M.D., Dunedin, New Zealand.

1894 †Fischer, E. C., M.D., Cairo, Egypt.

1904 Fisher, Major J., I.M.S., c/o Messrs. Grindlay and Co., 54, Parliament Street, S.W.

1895 Fisher, J. Herbert, M.B., 83, Wimpole Street, W. (C. 1904-7; 1911-12. S. 1907-10.)

1912 Flaxman, Samuel C. R. (Address uncommunicated.)

1917 Fleming, Norman B. R., M.B., Royal Westminster Ophthalmic Hospital.

1889 Flemming, Percy, M.D., 70, Harley Street, W. (V.-P. 1911–13; C. 1901-4.)

1892 Folker, Herbert Henry, Hanley, Staffordshire.

1886 Ford, A. Vernon, 8, Outram Road, Southsea.

O.M. Frost, W. Adams, Shepherd's Well, Forest Row, Sussex. (L. 1900-6. V.-P. 1906-9.)

1895 Galloway, A. Rudolf, M.B., 250, Union Street, Aberdeen.

1887 †Gardner, John J., M.D., 128, Bishop Street, Montreal, Canada.

1913 Ghaswala, K. S., 3, Churchgate Street, Fort Bombay, India; and c/o Sir Cowasji Jehangir Ophthalmic Hospital, Byculla, Bombay.

1912 Gibb, H. P., M.B., 91, Harley Street, W.

1889 Gibbs, Alfred N. G., 52, Whiteladies Road, Clifton.

1902 †Gibson, J. Lockhart, M.D., Wickham Terrace, Brisbane, Australia.

O.M. Glascott, C. E., M.D., Rosemullion, Budleigh Salterton, Devon. (C. 1896-9. V.-P. 1902-5.)

1885 †Godfray, Alfred Charles, St. Heliers House, St. Heliers, Jersey.

1899 Goldsmith, G. Harvey, M.B., Bedford.

1907 Goulden, Charles Bernard, M.D., 5, Union Street, Oldham.

ELECTED

1904 Gowans, Thos., M.B., 4, Abbotsford Terrace, Newcastle-on-Tyne.

1897 Granger, F. M., 18, Nicholas Street, Chester.

1895 †Grant, H. Y., M.D., 414, Delaware Avenue, Buffalo, U.S.A.

1895 †Gray, James, M.D., 119, Robert Street, Toronto, Canada.

1887 Green, Edwin Collier, 27, Friar Gate, Derby. (C. 1903-4.)

1903 Greene, Arthur, M.D., 4, Theatre Street, Norwich.

1911 Greeves, Reginald A., M.B., 14, Harley Street, W.

1895 Griffin, W. Watson, M.B., 68, Brunswick Place, Hove, Brighton.

1885 *Griffith, A. Hill, M.D., 17, St. John Street, Manchester. (V.-P. 1905-8. C. 1893-6.)

1910 Griffith, Arthur Donald, M.B., 3, Upper Wimpole Street, W.

1894 Grimsdale, Harold, M.B., 3, Harley Place, Marylebone Road, N.W. (C. 1907-10.)

1910 Gròsz, Prof. E. von (F.), 5, Reviczhy Ter., Buda-Pest, Hungary.

1899 Gruber, Rudolph, M.D., 81, Harley Street, W.

1908 Gunning, C. J. H., 6, Hertford Street, Mayfair, W.

1898 Guthrie, Leonard G., M.D., 15, Upper Berkeley Street, W.

1910 Haab, Prof. O., 41, Pelikanstrasse, Zurich, Switzerland.

1889 Hailes, C. D. G., M.D., Hawkesdale, 27, Alma Road, Clifton, Bristol.

1885 †Haines, Humphrey, Auckland, New Zealand.

1902 †Halliday, J. C., M.B., Macquarie Street, Sydney, N.S.W.

1900 Hallidie, Andrew, M.B., 7, Devonshire Place, Eastbourne.

1896 Hamilton, Robert J., 82, Rodney Street, Liverpool.

1878 †Hamilton, T. K., M.D., Wakefield Street, Adelaide, South Australia.

ELECTED

1912 Hanafin, Capt. P. J., R.A.M.C. (Address uncommunicated.)

1901 Hancock, Edw. D., Merrow Grange, Guildford.

1908 Hanna, Henry, M.B., 57, University Road, Belfast.

1901 Hanson, Reginald E., M.B., 20, Kensington Park Gardens, W.

1900 Harman, N. B., M.B., 108, Harley Street, W.

1902 Harries-Jones, E., M.D., 16, Castilian Street, Northampton.

1910 Harston, George M., M.D., Hong Kong.

1882 *Hartridge, Gustavus, 12, Wimpole Street, W. (C. 1889-92, 1895-8. S. 1892-5. V.-P. 1910-11.)

1905 †Harty, G. W., M.B., Ch.B., Wellington, New Zealand.

1899 Hawkes, C. S., Glencairn, Wickham Terrace, Brisbane, Queensland.

1901 Hawthorne, C. O., M.D., 63, Harley Street, W.

1908 Hay, Percival J., M.D., 350, Glossop Road, Sheffield.

1892 Haydon, Frank, Apothecaries' Hall, Water Lane, Blackfriars, E.C.

1899 Henderson, Edward E., M.B., 6, Lower Berkeley Street, Portman Square, W.

1903 Henderson, Thomson, M.D., 15, The Ropewalk, Nottingham.

1896 Henry, R. Wallace, M.D., 6, Market Street, Leicester.

1905 Hepburn, Malcolm Langton, M.D., 111, Harley Street, W.

1897 Herbert, Lt.-Col. Herbert, I.M.S., 9, Regent Street, Nottingham. (C. 1911–13.)

1887 *Hern, John, M.D., Semmercote, Darlington.

1910 Heron, David, R.A.M.C. Mess, Grosvenor Road, S.W.

1910 von Hess, Prof. Carl, 1, Pleichenglacisstrasse, Würzburg, Bavaria.

1905 Hewkley, F., The Limes, St. James, Hatcham, S.E.

1900 Heyliger, E. D. Scott, M.B., C.M., 89, Preston New Road, Blackburn.

ELECTED

1895 Hickman, H. R. Belcher, M.B., The Hollies, Wanstead, N.E.

O.M. Higgens, Charles, 52, Brook Street, W. (C. 1880-3, V.-P. 1901-4.)

1913 Hill, Rowland, M.D., 7, University Road, Belfast.

1916 Hine, Montague, M.D., 30, Weymouth Street, W.

1888 *Hinnell, J. S., M.D., 62, Garland Street, Bury St. Edmunds.

1905 Hird, R. B., M.D., 52, Newhall Street, Birmingham.

1899 Hobday, James, M.B., Beverley House, Colney Hatch Lane, N.

1897 Hogg, G. H., M.D., 37, Brisbane Street, Launceston, Tasmania.

1911 Holmes, Lt.-Col. Gordon M., C.M.G., M.D., (C.), R.A.M.C., B.E.F., France. (S. 1912–15.)

1889 Holthouse, Edwin H., M.B., 35, Nottingham Place, W.

1905 Hosford, J. S., 7, Chandos Street, W.

1893 †Howe, Lucien, 520, Delaware Avenue, Buffalo, N.Y., U.S.A.

1908 Hudson, A. C., M.D., 50, Queen Anne Street, W.

1889 †Hughes, Samuel H., 173, Macquarie Street, Sydney, New South Wales.

1893 †Hughes, Wilfrid Kent, M.B., 102, Collins Street, Melbourne.

1908 Hunter, George, 31, Bridge Street, Inverness.

1901 Inman, Wm., M.B., 55, Elm Grove, Southsea.

1883 †Jackson, James, M.D., Collins Street East, Melbourne, Australia.

1898 James, George Brooksbank, 5, Harley Street, W. (C. 1911–13.)

1908 James, Robert Rutson, 6, Lower Berkeley Street, W.

1905 Jaques, R., 20, Athenæum Street, Plymouth.

1883 †Jenkins, E. J., M.D., Nepean Towers, Douglass Park, Sydney, N.S.W., Australia.

ELECTED

1908 Jeremy, H. Rowe, 8, Chingford Road, Walthamstow, Essex.

1910 Jewell, William H., M.D., 27, Queen Anne Street, W.

1882 Johnson, G. Lindsay, M.D., 5 and 6, Castle Mansions, Eloff Street, Johannesburg, S.A.

1888 †Johnston, Geo. D., Georgia Street, Vancouver, British Columbia.

1898 *Jones, George, M.B., 8, Church Terrace, Lee, S.E.

1897 Jones, Hugh E., 19, Rodney Street, Liverpool.

1894 †Jones, R. H., M.B., B.S., 209, Macquarie Street, Sydney, New South Wales.

1914 Joseph, Hugh Percival, The Hospital Bungalow, Gallé. Ceylon.

1917 Joynt, Ivor William, Royal London Ophthalmic Hospital, E.C.

1905 Juler, F. A., M.B., 24, Cavendish Square, W.

O.M. Juler, H. E., 24, Cavendish Square, W. (C. 1886-9. V.-P. 1900-2.)

1914 Karani, N. N., "Eye Clinic," near Edward Theatre, 18, Kalbadevi Road, Bombay.

1899 Keeling, G. S., M.D., Attleborough, Norfolk.

1900 †Kelsall, H. T., M.D., 1, Devonshire Terrace, Perth, W. Australia.

1898 †Kendall, H. W. Martindale, Wellington, New Zealand.

1888 †Kenny, Augustus Leo, M.B., 87, Collins Street, Melbourne, Victoria, Australia.

1913 Killen, W. M., M.D., 34, Clifton Street, Belfast.

1904 Killick, Chas., M.D., Trinity House, Maidstone.

1906 Kilkelly, Major P. P., I.M.S., c/o Messrs. H. S. King and Co., Pall Mall, S.W.

1895 Knaggs, Robert Lawford, M.D., 27, Park Square, Leeds.

1881 †Knaggs, S. T., M.D., Wellington, Bondi Road, Bondi, Sydney, N.S.W.

1910 Knapp, Arnold, M.D., 10, East 54th Street, New York, U.S.A.

ELECTED

1911 Lang, B. T., B.C., 22, Cavendish Square, W.

O.M. Lang, William, 22, Cavendish Square, W. (C. 1886-9. V.-P. 1900-3. (*Acting Pres.* 1903.)

O.M. Lawford, J. B., M.D., 99, Harley Street, Cavendish Square, W. (C. 1886-9; 1898-1901. S. 1895-8. V.-P. 1905; 1913-16. T. 1905-11. *Pres.* 1911-13.)

1889. *Laws, Wm. G., M.B., 3, East Circus Street, Nottingham. (C. 1909-11.)

1896. Lawson, Arnold, M.D. (V.-P.), 12, Harley Street, W. (C. 1909-1911.)

1895 †Lea, J. Augustus, M.B., Grahamstown, Cape Province, S. Africa.

1885† Le Cronier, Hardwick, St. Heliers, Jersey.

1917 Leask, H. L. G., M.D., 70, Dixon Avenue, Crosshill, Glasgow.

O.M. Lediard, H. A., M.D., 26, Lowther Street, Carlisle. (C. 1900-1.)

1912 Lee, H., M.B., 6, Park Square, Leeds.

1904. †Lee, W. A., Lt.-Col. I.M.S., 44, Primrose Hill Road, N.W.

1911 Letchworth, T. W., M.B., 68, Claremont Road, Surbiton.

1903 Levy, A., M.D., 67, Wimpole Street, W.

1907 Lindsay, W. J., M.D., 84, Herne Hill, S.E.

1896 Lister, Col. W. T., C.M.G., M.B., A.M.S., Hotel Meurice, Boulogne, B.E.F. (C. 1907-10.)

1902 Little, Andrew, M.B., 114, Manningham Lane, Bradford, Yorks.

1892 Lodge, Samuel, jun., M.D., 28, Manor Road, Bradford.

1911 Loosely, Alfred E. A., M.B., 25, New Cavendish Street, W.

1903 †Lückhoff, James, M.D., Rhodes Buildings, St. George's Street, Capetown.

1883 Lunn, J. R., The Haven, Mill Road, Worthing. (C. 1892-5.)

1899 Lyle H. Willoughby, M.D., Speldhurst, Elmfield Road, Bromley, Kent.

ELECTED

1900 MacCallan, Arthur Ferguson, M.B., Public Health Department, Cairo, Egypt.

1913 McCready, W., M.B., 2, University Square, Belfast.

1905 Macdonald, J. G., Imperial Chambers, Dee Street, Invercargill, New Zealand.

1890 *MacGillivray, Angus, M.D., 23, South Tay Street, Dundee. (C. 1902-5.)

1902 Mackay, D. Matheson, M.D., 15, Albion Street, Hull.

1888 *Mackay, George, M.D., 20, Drumsheugh Gardens, Edinburgh. (C. 1900-2. V.-P. 1906-9.)

1889 †Mackenzie, F. Wallace, M.B., 139, Upper Willis Street, Wellington, New Zealand

1895 McKenzie, H. V., M.D., Elmbank, Abbey Road, Torquay.

1889 MacLehose, Norman M., M.B., 18, Harley Street, W. (C. 1902-5.)

1897 †Maclennan, Duncan N., M.D., 126, Bloor Street West, Toronto.

1892 †MacLeod, Charles G., M.D., 157, Macquarie Street, Sydney, N.S.W.

1902 McMullen, W. H., M.B., 4, Chandos Street, Cavendish Square, W. (C. 1914-17.)

1911 McNabb, Henry H., M.D., 8a, St. John Street, Manchester.

1912 McPherson, Major, I.M.S., c/o Ophthalmic Hospital, Bombay, India.

1911 MacQueen, J. F. (Address uncommunicated.)

1899 Maddox, Ernest E., M.D., Glenartney, Poole Hill, Bournemouth. (C. 1904-6.)

1883 †Maher, W. Odillo, M.D., Craignish, 185, Macquarie Street, Sydney, N.S.W.

1899 †Manché, Charles, B.A., M.D., 33, S^da. Alessandro, Valletta, Malta.

1901 †Manning, Leslie S., M.B., Christchurch, New Zealand.

1911 Manson, W. H., M.D., 5, Clifton Place, Glasgow, W.

1904 Markus, Charles, M.D., 28, Wimpole Street, W.

ELECTED

1883 †Marlow, Frank William, M.D., 200, Highland Street, Syracuse, N.Y., U.S.A.

1916 Marquez, M., Dr., Puerta del Sol, 13, Madrid, Spain.

1892 Marshall, Charles Devereux, 112, Harley Street, W. (C. 1903-4. S. 1904-7. C. 1907-10.)

1907 Marshall, James Cole, M.D., 36, Albion Street, Hyde Park, W.

1888 †Martin, Albert, M.D., Wellington, New Zealand.

1905 Massey, A. Yale, M.D., 24, Tavistock Square, W.C.

1916 Maxted, George, M.D., 14, Thorpe Mansions, Norwich.

1914 Maxwell, Miss E. M., M.B., 19, Lower Baggot Street, Dublin.

1904 May, H. J., M.B., B.C., 21, The Avenue, Southampton.

1893 Maynard, Frederic Pinsent, Lt.-Col., I.M.S., 13, Harington Street, Calcutta.

1902 Mayou, Stephen (S.), 30, Cavendish Square, W. (C. 1912–15.)

1912 Mead, John Clarke, M.B., 55, High Street, Lowestoft.

1901 Menzies, J. A., M.B., 9, Castle Hill Avenue, Folkestone.

1897 Miller, G. Victor, M.D., 2, Barrington Crescent, Yarm Lane, Stockton-on-Tees.

1881 †Milles, W. Jennings. (Address uncommunicated.)

1897 †Minnes, Robert Stanley, M.D., 127, Metcalfe Street, Ottawa, Ontario.

1911 Mitchell, Leonard J. C., 4, Collins Street, Melbourne.

1896 Mooney, Herbert C., M.B., 33, Fitzwilliam Place, Dublin.

1908 Moore, Robert F., M.B., 91, Harley Street, W.

1909 Morrell, Reginald A., 13, Harley Street, W.

O.M. Morton, A. Stanford, M.B., 133, Harley Street, W. (C. 1886-9. V.-P. 1902-5.)

1896 Mott, F. W., M.D., F.R.S., 25, Nottingham Place, W. (V.-P. 1904–7.)

ELECTED

1908 Moxon, Frank, M.B., B.S., 56, Seymour Street, Portman Square, W.

1916 Mulholland, P. J. H., 5, University Terrace, Belfast.

1909 Naylor, W. Roebuck, 14, Carr Road, Nelson, Lancs.

1909 Nell, Andreas, Victoria Eye and Ear Hospital, Colombo, Ceylon.

1881 Nicholson, A., M.B., 30, Brunswick Square, Brighton.

1910 Nordenson, Dr. Erik, 10, Wasagatan, Stockholm, Sweden.

1912 Norman, Alfred Clarence, M.D., Durham County and Sunderland Eye Infirmary, Sunderland.

1895 *Ogilvie, F. Menteith, M.B., The Shrubbery, 72, Woodstock Road, Oxford. (C. 1910-12.)

1889 †O'Kinealy, Frederick, Lt.-Col., I.M.S., c/o Messrs. King, Hamilton and Co., 7, Hare Street, Calcutta.

1899 Oldmeadow, Lloyd J. H., M.D., Kineton, Warwickshire.

1911 Oliver, Capt. M. W. B., M.B., R.A.M.C., 24th General Hospital, B.E.F., France.

1914 Oliver, George H., 284, Manningham Lane, Bradford.

1899 Ormond, Arthur Wm. 7, Devonshire Place, W. (C. 1910-12).

1892 †Orr, Andrew William, M.D., 71, Wickham Terrace, Brisbane.

1890 †Osborne, A. B., M.D., 46, McNab Street South, Hamilton, Canada.

O.M. Owen, D. C. Lloyd, M.D., 41, Newhall Street, Birmingham. (V.-P. 1891-4.)

1905 Owen, S. A., M.D., 15, Queen Anne Street, W.

1890 †Palmer, L. Loran, M.D. (Address uncommunicated.)

1894 *Parker, Herbert, Saragossa House, New Street, Henley-on-Thames.

1899 Parker, Herbert George, 13, Chorley New Road, Bolton.

1900 Parsons, John Herbert, M.B., D.Sc., 54, Queen Anne Street, W. (C. 1909-12).

ELECTED

1914. Pascheff, Constantin, M.D., 151, Rue Rakowsky, Sofia, Bulgaria.

1887 †Patel, D. H., Bai Hirabai, B. J. Charitable Dispensary Tardeo, Bombay.

1907 Paterson, James V., M.B., 11, Melville Street, Edinburgh.

1900 †Patkar, Bhagvant Sakharam, Carnac Road, Kalkadeir Post, Bombay, India.

1902 Paton, Leslie, M.B., 29, Harley Street, W. (C. 1912–15.)

1888 Percival, Archibald Stanley, M.B., B.Ch., 25, Ellison Place, Newcastle-on-Tyne. (C. 1906-9.)

1891 Perry, Sir A., M.D., Surgeon-Major, Principal Civil Medical Officer, Colombo, Ceylon.

1889 †Perry, Francis F. (Address uncommunicated.)

1895 Pickard, Ransom, M.D., 31, East Southernhay, Exeter.

1916 Pike, N., M.D., 10, York Terrace, St. George's Road, Cheltenham.

1910 Pisani, Lt.-Col. L. J., I.M.S., 49, Wimpole Street, W.

1900 †Pockley, Francis Antill, M.D., 227, Macquarie Street, Sydney, N.S.W.

1903 Pollock, W. B. Inglis, M.D., 21, Woodside Place, Charing Cross, Glasgow.

1896 *Pooley, G. H., 70, Hanover Street, Glossop Road, Sheffield.

1894 †Pope, R. J., M.D., Box 497, G.P.O., Sydney, N.S.W.

1900 Pope, Thomas Henry, M.D., 39, Price's Avenue, Margate.

1902 Potter, Bernard E., M.B., 58, Park Street, W.

1903 *Potts, George, Bower Cottage, Tonbridge Road, Maidstone.

1899 Price, Henry J., Maldon, Essex.

1882 Prichard, Arthur William, 6, Rodney Place, Clifton, Bristol.

ELECTED

1908 Pridmore, Walter G., Major I.M.S., c/o Grindlay and Co., 54, Parliament Street, S.W.

1903 Pritchard, Eric L., M.D., 70, Fairhazel Gardens, N.W.

1892 Pronger, Charles Ernest, Litchdon, Harrogate.

1909 Prowse, S. Willis, M.D., 706, Union Bank Buildings, Winnipeg, Canada.

O.M. Purves, W. Laidlaw, 20, Stratford Place, Oxford Street, W.

1911 Quick, Hamilton E., M.B., 51, Bryn Road, Swansea.

1889 Ramsay, A. Maitland M.D. (V.-P.), 15, Woodside Place, Glasgow. (C. 1909-11.)

1899 Read, E. I., Government Medical Officer, Port of Spain, Trinidad, West Indies.

1881 †Reeve, R. A., M.D., 22, Shuter Street, Toronto, Canada. (V.-P. 1907-10.)

1891 Reynolds, Austin Edward, 35, Finsbury Square, E.C.

1913 Riad, M., Kasr el Aino Hospital, Cairo, Egypt.

1897 Richmond, R., M.D., 29, Lingfield Road, Wimbledon, S.W.

1892 Ridley, Nicholas C., M.B., 27, Horse Fair Street, Leicester.

1914 Roberts, Miss Adeline Mary, M.D., 4, Nottingham Place, W.

1885 *Roberts, Edward, Penywern, Aberystwith, Cardiganshire.

1896 †Roberts, J. R., Major, M.B., c/o Messrs. King, King and Co., Bombay.

1891 Robertson-Fullarton, Archibald Louis, M.B., C.M. Kilmichael, Brodrick, Isle of Arran.

O.M. Rockliffe, W. C., M.D., 17, Charlotte Street, Hull. (C. 1892-5. V.-P. 1900-3.)

1912 Rodger, Douglas, M.B., Dept. of Public Instruction, Brisbane, Queensland.

1898 Roe, Arthur Legge, 43, Pryme Street, Hull.

ELECTED

1898 Roll, G. W., M.B., B.C., 7, Upper Wimpole Street, W. (C. 1910-12.)

1890 Rolston, John R., 14, The Crescent, Plymouth.

1891 Roper, Arthur Charles, The Shrubbery, Exeter.

1882 †Roth, Reuter E., D.S.O., 42, College Street, Hyde Park, Sydney, New South Wales.

1893 *Rowan, John, M.B., 10, Woodside Crescent, Charing Cross, Glasgow.

1899 *Roxburgh, A. B., M.B., 3, Manchester Square, W.

1881 †Rudall, J. T., 61, Spring Street, Melbourne, Australia.

1895 Russell, J. S. Risien, M.D., 44, Wimpole Street, W. (C. 1900–3. S. 1903-6. C. 1906-9.)

1903 Rutherford, A. Freer, M.B., 19, Abbey Road, Barrow-in-Furness.

1900 †Sager, D. S., M.D., Brantford, Ontario, Canada.

1884 *Sandford, Arthur W., M.D., 13, St. Patrick's Place, Cork. (C. 1896-9. V.-P. 1902-5.)

1907 Scott, G. Affleck, M.B., Kingsland House, Ballarat, Victoria.

1888 Scott, Kenneth, M.D., 7, Manchester Square, W.

1892 Shannon, John Rowlands, M.D., 17, East 38th Street, New York City, U.S.A.

O.M. Sharkey, Sir S. J., M.D., 24a, Portland Place, W. (S. 1885-8. C. 1888-91. V.-P. 1900-2.)

1883 Shears, Charles H. B., 19, Upper Duke Street, Rodney Street, Liverpool. (C. 1900–3.)

1911 Sheedy, Thos. (Address uncommunicated.)

1899 Sinclair, A. H. H, M.B. (C.), 3, Charlotte Square, Edinburgh.

1891 Sinclair, Walter William, M.B., 3, Arcade Street, Ipswich. (C. 1909-1912.)

1912 Smith, D. Priestley, M.B., 52, Frederick Road, Edgbaston, Birmingham.

1889 Smith, John, M.D., Brycehall Kirkcaldy.

ELECTED

O.M. Smith, Priestley, M.B., M.Sc., 95, Cornwall Street, Birmingham. (V.-P. 1887-90; 1898-1901; 1907-10. C. 1883-6. *Pres.* 1905-7.)

1907 Smith, W. Harvey, M.D., 173, Portage Ave., E. Winnipeg, Canada.

1903 Smyth, Ernest J., M.B.. Sevington, Epsom Road, Guildford.

1911 Snell, H. Cecil, Moor Lodge, Sheffield.

1910 Snellen, Prof. H. Biltstraat, Utrecht, Holland.

1901 Snowball, Thomas, M.B., 83, Bank Parade, Burnley.

1889 Spencer, Matthew H., M.D., 92, Oxford Gardens, North Kensington, W.

1889 Spicer, Wm. T. Holmes, M.B. (L.), 5, Manchester Square, W. (C. 1900-2. L. 1907-12. V.-P. 1910-12.)

1905 Sprawson, F. C., M.B., 7, Imperial Terrace, Claremont Park, Blackpool.

1897 Square, James Elliot, 22, Portland Square, Plymouth.

1916 Stack, E. H., M.B., B.C., Arvalee, Clifton Down Road, Bristol.

1895 †Stamberg, A. C., M.B., 5, Windsor Crescent, St. Heliers, Jersey, Channel Islands.

1886 Stephenson, Sydney, M.B. (V.-P.), 33, Welbeck Street, W. (C. 1898-1901, 1904-7. S. 1901-4.)

1896 Stevenson, Edgar, M.D., 39, Rodney Street, Liverpool.

1904 Stevenson, John Simpson, M.B., Royal Exchange Buildings, Cathedral Square, Christchurch, New Zealand.

1905 Steward, E. S., 10, Princes' Square, Harrogate.

1913 Stewart, John Barbour, M.B., 20, Charing Cross Mansions, Glasgow.

1893 Stirling, Alexander Williamson, M.D., Empire Buildings, Atlanta, Georgia.

1887 †Stirling, J. W., M.B., 255, Mountain Street, Montreal, Canada.

O.M. Story, J. B., M.B., 6, Merrion Square North, Dublin. (C. 1885-8. V.-P. 1894-7.)

ELECTED

O.M. †Sturge, W. A., M.D., Icklingham Hall, Mildenhall, Suffolk.

1910 Sulzer, Dr., 3, Rue Troyon, Place de l'Etoile, Paris.

1904 Sykes, Walter, 31, Winckley Square, Preston.

1888 *Sym, William George, M.D. 12, Alva Street, Edinburgh. (C. 1906-9.)

1883 †Symons, Mark Johnston, M.D., North Terrace, Adelaide, South Australia.

1907 Tampi, K. R., Trivandrum, South India.

O.M. Tay, Waren, 61, Oakfield Road, West Croydon. (C. 1880-2.)

1903 Taylor, H. H., 36, Brunswick Square, Hove, Brighton.

1899 Taylor, Inglis, M.B., 31, Cavendish Square, W.

1891 Taylor, James, M.D., (T.), 49, Welbeck Street, W. (V.-P. 1910-11. C. 1894-7, 1900–3. S. 1897-1900.)

1889 Taylor, S. J., M.B., 44, Prince of Wales' Road, Norwich. (C. 1904-7.)

1914 Terson, Albert, M.D., 47*bis*, Boulevard des Invalides, Paris.

1900 Thomas, Frank G., M.B., 22, Walter Road, Swansea.

1903 Thomas, R. Russell, 40, St. Andrew's Crescent, Cardiff.

1895 Thompson, A. Hugh, M.D., 36, Weymouth Street, W. (C. 1910-12.)

1895 Thompson, George W., M.B., 80, Harley Street, W.

1895 *Thompson, Robert, M.D., B.S., Bunya Bunya, Wickham Terrace, Brisbane, Queensland.

1911 Thomson, Eric A., M.B., British Ophthalmic Hospital, Jerusalem.

1905 Thomson, H. Wright, M.D., 3, Sandyford Place, Glasgow.

1898 Thomson, W. Ernest, M.D., 2, Somerset Place, Glasgow.

1883 †Tobin, William, c/o Messrs. John Tobin and Co., Halifax, Nova Scotia, Canada.

1900 Tomlinson, John H., Tudor Lodge, Egham, Surrey.

ELECTED

1904 †Tooke, Frederick T., M.D., 1, MacGregor Street, Montreal, Canada.

1895 Toombs, Herbert George, 193, Cromwell Mansions, Cromwell Road, S.W.

O.M. Tosswill, L. H., M.B., 7, Barnfield Crescent, Southernhay, Exeter. (C. 1896-9.)

1904 Townsend, T. H D., M.B., 14, St. Patrick's Hill, Cork.

1912 Traquair, H. Moss, M.D., 46, Melville Street, Edinburgh.

1905 Trench, F. P., M.B., 48, Chester Terrace, Eaton Square, S.W.

1902 †Trilokekar, V. S., 308, Lohar Street, Bombay, India.

1917 Tuckett, Ivor Lloyd, M.D., Punchardon William, near Letchworth, Herts.

O.M. Tweedy, Sir John, LL.D., 100, Harley Street, W. (*Pres.* 1903-5. C. 1884-7. V.-P. 1891-4, 1905-8.)

1898 Tyrrell, F. Astley-Cooper, M.B., 27, Queen Anne Street, W.

1883 Uhthoff, J. C., M.D., Wavertree House, Furze Hill. Brighton. (C. 1905-8. V.-P. 1908-12.)

1910 Uhthoff, Prof. W., 16 ap. Schweidnitzer Stadtgraben, Breslau.

1894 Usher, C. H., M.B. 3, Bon Accord Square, Aberdeen. (C. 1909-1912.)

1916 Valentine, John A., M.D., 65, Devonshire Avenue, Southsea.

1913 Viner, Geoffrey, M.D., 15, Devonshire Place, W.

1910 Wagenmann, Prof., Bergstrasse 80, Heidelberg, Germany.

1888 Walker, Cyril H., M.B., 8, Oakfield Road, Clifton, Bristol. (C. 1907-10),

1892 Walker, H. Secker, 45, Park Square, Leeds. (C. 1903-6.)

1916 Wallace, William, M.D., 11, Ladbroke Grove, W.

ELECTED

1916 Wallis, G. F. C., M.D., 1, Price's Avenue, Cliftonville, Margate.

1900 Wardale, John D., M.B., Carlton Villa West, Jesmond Road, Newcastle-on-Tyne.

1893 Warren, H. Guy S., 201, Macquarie Street, Sydney, Australia.

1914 Weeks, John Elmer, M.D., D.Sc., 46, East 57th Street, New York City, U.S.A.

1910 †Weihen, A. Wallace, M.D. (Address uncommunicated.)

1887 Wells, Arthur P. L., M.B., 48, Belzize Square, Hampstead, N.W. (C. 1896-9.)

1885 Werner, Louis, M.B., 31, Merrion Square North, Dublin. (C. 1902-5.)

1908 Wharton, John, M.D., 21, St. John Street, Manchester.

O.M. *Wherry, G. E., M.B., Corpus Buildings, Cambridge (C. 1897-1900.)

1895 Whitehead, Arthur Longley, M.B., 31, Park Square, Leeds. (C. 1912–15.)

O.M. Williams, R., 125, High Street, Bangor, North Wales (V.-P. 1896-9.)

1888 †Willis, C. Fancourt, C.B., M.D., Col., I.M.S., Frith Manor, East Grinstead.

1914 Wilson, James Alexander, M.D., 4, Central Avenue, Cambuslang, Glasgow.

1911 Wilson, S. A. Kinnier, M.D. (S.), 14, Harley Street, W.

1900 Wood, C. G. Russ, 12, St. John's Hill, Shrewsbury.

1889 Wood, David J., M.B., National Mutual Building, Church Square, Cape Town, South Africa.

1903 Wood, Percival, The Grange, Crawley.

1909 Wood, R. Moorsom, 32, Beaumont Street, W.

O.M.* Woodhead, G. Sims, M.D., LL.D., Dysart House, Luard Road, Cambridge. (C. 1894-5. V.-P. 1905-8.)

ELECTED.

1899 *Worth, Claud, 138, Harley Street, W.

1910 Worton, A. S., M.D., 71, Harley Street, W.

1890 *Wray, Chas., Bank Chambers, North End, Croydon.

1898 †Wright, Edward Wm., M.D., 115, Montague Street, Brooklyn, New York.

1908 Wright, F. R. Elliston, M.B., Braunton, North Devon.

1896 Yarr, Col. M. T., C.B., R.A.M.C., Army and Navy Club, Pall Mall, S.W.

1910 Yearsley, J. Herbert, "Giffnock," 6, Christchurch Road, Bournemouth.

1917 Young, George, M.D., Cambridge Road, Colchester.

1912 Zorab, Arthur, M.B., 21, Carlton Crescent, Southampton.

RULES.

1. Object of the Society.—The object of the Society is the cultivation and promotion of Ophthalmology.

2. Meetings.—The Society shall hold a meeting each year the sittings shall be held on two or more consecutive days. On each day there shall be usually two sittings. The date and place of meeting shall be chosen by the Council.

3. Constitution.—The Society shall consist of Ordinary and Honorary members. All medical practitioners whose qualifications are satisfactory to the Council of the Society shall be eligible as Ordinary members. Other Ophthalmological Societies in the British Empire, approved by the Council, may become affiliated.

4. Officers of the Society.—The officers of the Society shall consist of a President, four Vice-Presidents, a Treasurer, two Secretaries and six other members, who together with Presidents of affiliated Societies (*ex-officio*) shall form the Council and manage the Society's affairs.

5. Election of Members.—Every applicant for admission to the Society shall send to one of the Secretaries a form signed by at least three members, who have personal knowledge of the applicant, or by the President and Secretary of an affiliated Society. Such application shall be submitted to the Council, and if approved the applicant shall forthwith be admitted a member.

6. Honorary Members.—The Council shall have the power of electing as Honorary members men of distinguished eminence in Ophthalmology, or in the sciences bearing upon it.

7. Subscriptions. — The Annual Subscription shall be £1 1*s*. 0*d*. payable in the month of October. No member whose subscription is in arrear shall be entitled to attend the meeting. Any member whose subscription is twelve months in arrear shall cease to be a member of the Society.

8. Election of Officers.—The Officers of the Society shall be elected yearly by Ballot at a Business sitting held during the meeting. A balloting list of the names recommended by the Council for election shall be posted to each member two weeks previously. At the Business sitting twelve shall form a quorum.

9. Duration of Office.—The President shall not hold office for a period of more than two consecutive years. No other member of Council, except the Treasurer, shall hold the same office for more than three consecutive years.

10. President and Vice-Presidents.—The President shall regulate all the proceedings of the Society and Council, state and put questions, interpret the application of the laws, and decide any doubtful points. He shall check irregularities and enforce the observance of the laws. He shall sign the minutes of General and Council meetings. In the absence of the President one of the Vice-Presidents, the Treasurer, or some other member chosen by the meeting, shall perform his duties.

11. Secretaries.—The Secretaries shall manage all correspondence, and shall keep the Minutes of the Society and Council. They shall notify to new members their election. They shall arrange with the President the order of proceedings at the meetings. They shall have charge of and shall keep a register of all papers communicated, and shall be the Editors of the 'Transactions.'

12. Treasurer.—The Treasurer shall receive all moneys due to the Society, and make all payments ordered by the Council, keeping an account of all such receipts and payments. The accounts shall be audited once a year and presented to the Society at the Annual Business Meeting. Two members of the Society shall be nominated by the President to audit the Treasurer's accounts.

13. Meetings of Council.—The Council shall meet as frequently as necessary during the Annual Meeting and at other times if specially convened. Five shall form a quorum. The President shall have a casting vote in addition to his ordinary vote. The Council shall decide upon all questions relating to the reception of communications, and shall have power to submit any paper to referees.

14. Vacancies of Offices.—The Council shall have the power of filling up any vacancies which may occur in any of the offices of the Society between one Annual Meeting and the next.

15. 'Transactions.'—The Society shall publish a yearly volume of 'Transactions' as soon as possible after the Annual Meeting (*vide* Bye Law 1 concerning communications). There shall be an Editorial Committee consisting of the President, the Secretaries and two other members of Council, together with the Secretaries of affiliated Societies. A copy of the 'Transactions' shall be sent to every member whose subscription has been duly paid; and to each Honorary member.

16. Publication of Papers and Discussion.—No paper shall be accepted if it has been published elsewhere. No paper shall be read before the Society unless a copy of it has been sent to the Secretaries at least four weeks before the meeting, together with an abstract suitable for immediate publication in the journals. When possible, notice should be given relating to card cases, and a short abstract of all papers to be read before the Society shall be distributed to all members before the meeting. No report of the meetings of the Society may be published by members or others without the sanction of the Council.

17. Notice of Meeting.—Three months before the date of the Annual Meeting a preliminary notice of the meeting shall be sent to every member.

18. Visitors.—Any member of the medical profession may attend a meeting of the Society (except the Annual Business Meeting) on being introduced by a member of the Society.

The Library has now been transferred to the premises of the Royal Society of Medicine, 1, Wimpole Street.

LIBRARY RULES.

1. The Library shall be open at the same hours as that of the Royal Society of Medicine, viz. from 11 a.m. to 6.30 p.m. daily, except Sundays.

2. Members will be entitled to read the books at 1, Wimpole Street, between these hours, or to take them out on signing a receipt provided for that purpose.

3. A large number of periodicals will be accessible to members in the Library. The current number may not be taken out of the Library.

4. A book must be returned at the expiration of a fortnight if wanted by any other member. The Librarian will in such a case write to the member in whose name the book was taken out.

5. If the book be not returned within four days of such notice a fine of 6d. will be charged for each day that the book is retained beyond such days of grace.

6. Instruments and drawings cannot be taken out of the Library except by special permission.

7. A member taking out a book will be held responsible for its being returned in good condition.

8. Members of the Ophthalmological Society who are also Members of the Section of Ophthalmology of the Royal Society of Medicine can have the full use of the Fellows' Library on payment of an additional guinea instead of the usual subscription of two guineas.

ADAMS FROST COLLECTION OF LANTERN SLIDES.

1. The slides shall be in the custody of the Librarian.

2. They may be lent, for the purpose of teaching, to teachers in any recognised Medical School on application to the Librarian.

3. The regulations under which the slides may be borrowed, together with a list of the slides, shall be printed (*a*) in the *Transactions;* (*b*) in the Catalogue of the Library; (*c*) on leaflets which may be sent to enquirers.

4. Other slides may be added to the collection, but the numbering (in so far as it coincides with that of the figures in Frost's *Fundus Oculi*) shall be retained.

5. The following are the regulations under which the slides may be borrowed :

Regulations for the Loan of Lantern Slides.

1. Not more than 20 slides may be borrowed at one time.

2. Not less than three days' notice of the wish to borrow slides shall be given, and a list giving the numbers of the slides required shall be sent to the Librarian.

3. The borrower shall sign a receipt for the slides, which will be given back to him on their return.

4. The slides must be returned within three days, no other slides can be lent to the same borrower till this has been done.

5. Any slides broken, lost, or damaged shall be replaced or repaired at the expense of the borrower.

THE BOWMAN LECTURE.

Resolution of Council, September 18th, 1883.

"That in recognition of Mr. Bowman's distinguished scientific position in ophthalmology and other branches of Medicine, and in commemoration of his valuable services to the Ophthalmological Society, of which he was the first President, the Council shall each year, or periodically, nominate some person to deliver a lecture before the Society, to be called 'The Bowman Lecture,' which shall consist of a critical *résumé* of recent advances in ophthalmology or in such subject or subjects as the Council shall select, or of any original investigation, and shall be delivered at a special meeting of the Society held for the purpose, at which no other business shall be transacted."

EDWARD NETTLESHIP PRIZE.

On the occasion of Mr. Nettleship's retirement from practice in 1901 a fund was inaugurated by his friends and pupils, with the object of founding an Edward Nettleship Prize for the Encouragement of Scientific Ophthalmic Work, and at a meeting of the General Committee of the above fund on July 2nd, 1902, the following resolutions were adopted :

1. That the prize should be a Gold Medal for the encouragement of scientific ophthalmic work.

2. That the Council of the Ophthalmological Society of the United Kingdom be asked to undertake the custody of the Prize and Fund, and to appoint Trustees.

3. The award of the Medal shall be entrusted to three members of the Ophthalmological Society appointed by the Council, such members to be changed after each award.

4. That the Medal shall be awarded at such intervals as shall be determined upon.

5. That only British subjects be eligible to receive this Prize.

6. That, subject to the discretion of the said Council, the Prize shall be awarded—

(*a*) For the most valuable contribution to Ophthalmology during the three years immediately preceding or since the last award, or

(*b*) For the best work done on any subject previously selected and announced by the Council of the Ophthalmological Society.

7. That in the event of no work being found of sufficient merit an award shall not be made.

Resolution of Council, January 25th, 1906.

"It shall be lawful for the Trustees, with the consent of the Council of the Society, to expend any surplus arising from the non-award of the Medal in any one year, or accumulated during several years, in the purchase of works for the Library of the said Society, such works to be inscribed 'Purchased by the Nettleship Prize Fund'; or, with the consent of the said Council, to expend such surplus in any manner calculated to promote the objects of the Society as defined in the Rules."

BYE-LAWS CONCERNING COMMUNICATIONS.

1. The 'Transactions' shall consist of such communications made to the Society, or to affiliated Societies, by or through members, as may be deemed by the Council suitable for publication. Also of discussions of importance or interest arising out of such communications.

2. No communication to the Society shall occupy more than twenty minutes, and in the subsequent discussion of it no member shall speak more than once, or for more than ten minutes, without the special permission of the Chairman.

3. All communications accepted by the Society become the property of the Society.

4. Communications are admissible which may have been read elsewhere, provided they have not been published.

5. The cost of illustrations shall be borne by the Society so far as, in the opinion of the Council, is consistent with the state of its funds.

6. Reprints of papers may be obtained by authors at their own expense, by arrangement with the printer.

REGULATIONS CONCERNING THE EXHIBITION OF PATIENTS AND OF PATHOLOGICAL SPECIMENS BY CARD.

A. A card, provided by the Society, must be placed conveniently near the patient (unless it is undesirable that it should be read by the patient or friends), and on it must be clearly written an account of the case, *comprising all the particulars intended for publication.* The title only of the case will be announced by the President to the meeting, but the Exhibitor (or his representative) must be present at the meeting, and be willing to read the case and furnish additional details if called upon to do so; the length of such oral communications not to exceed five minutes.

B. Pathological Specimens may, at the discretion of the Exhibitor, be shown by card, and will then be subject to the above regulations. It is particularly to be noted that the description on the card must *comprise all the particulars intended for publication.*

LIST OF COMMUNICATIONS

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1916–17.

PAGE

I. PRESIDENTIAL ADDRESS.
By E. Treacher Collins 1

II. COLOUR-SENSE PHENOMENA AND SOME INFERENCES WHICH THEY SEEM TO SUGGEST. Being the Bowman Lecture.
By Sir George A. Berry 5

III. DISCUSSION ON CONCUSSION INJURIES OF THE VISUAL APPARATUS.
Opening Paper. By A. W. Ormond 60
Opening Paper. By S. A. Kinnier Wilson 92

IV. DISCUSSION ON THE EMPLOYMENT OF THE BLIND
Opening Paper. By Sir A. Pearson 133

V. CONGENITAL ABNORMALITIES.
1. An unusual case of opaque nerve-fibres.
By Leslie Buchanan 166

PAGE

2. Small tortuous veins on the optic disc in association with small vascular nævi of the skin of the eyelid and of the nasal septum.
By E. Treacher Collins 171
3. Unilateral melanosis of a segment of the iris combined with pigmented nævus of the lid margin and pigmented nævi in the skin of the face in a dark-complexioned subject. By A. C. Hudson 174
4. Congenital buphthalmos, with enlargement of the arteries of the retina. By H. Grimsdale 175

VI. INJURIES.

1. Protection of the eyes in warfare.
By R. R. Cruise 176
2. Pulsating exophthalmos as the result of a wound of the head. By M. S. Mayou 186

VII. DISEASES OF THE EYELID AND ORBIT.

1. Case of functional blepharospasm in an old man, causing temporary loss of sight.
By I. L. Tuckett 188
2. Papilloma of the conjunctiva. By M. L. Hepburn 190
3. Orbital suppuration. By M. S. Mayou 191

VIII. DISEASES OF THE CORNEA.

1. Case of Mooren's ulcer treated with a conjunctival flap. By F. A. C. Tyrrell 205

IX. DISEASES OF THE UVEAL TRACT.

1. A case of "uveo-parotitis" with irido-cycloplegia.
By George Mackay 208
2. Iridocyclitis of obscure origin.
By D. Leighton Davies 220

PAGE

3. Two cases of herpes zoster affecting the ciliary nerves. By M. S. Mayou 226
4. (?) Colloid nodules in the choroid in a girl. By M. S. Mayou 227
5. Leuco-sarcoma of choroid and epithelioma of lip, in the same patient. By J. A. Valentine 229

X. DISEASES OF THE RETINA AND OPTIC NERVE.

1. A case of detachment of the retina at the ora serrata. By A. Lawson 231
2. Symmetrical pigmented changes at the macula. By F. A. C. Tyrrell 237
3. Fundus lesions in cases of war nephritis. By J. Jameson Evans 238
4. Hereditary glioma of the retina. By A. Hill Griffith 242
5. Two cases of Leber's disease (hereditary optic atrophy), with X-ray examination of the sella turcica. By W. B. Inglis Pollock 247
6. A further case of Leber's disease, and two allied cases, associated with changes in the sella turcica. By J. Herbert Fisher 251

XI. DISEASES OF THE LENS.

1. The history of couching for cataract. By R. H. Elliot 264

XII. INTRA-OCULAR TENSION.

1. The sequelæ of hypotony. By E. Treacher Collins 281
2. Sclero-corneal trephining for hypertony: an experience of some two hundred and fifty operations. By J. Gray Clegg 308

PAGE

XIII. MISCELLANEOUS.

1. The evolution of the ophthalmoscope. By W. H. McMullen 324
2. The apocritic principle and the evolution of visual perceptions. By J. Herbert Parsons 333
3. The value of the ophthalmometer in estimating the refraction of the eye. By Ernest Clarke 343
4. Conditions affecting standards of vision in an army. By W. Wallace 349
5. Remarks upon advancement operations. By C. Worth 365
6. Functional spasm of accommodation. By Leslie Paton 370

XIV. APPENDIX AND MUSEUM SPECIMENS 381

Report of Council 383
Balance Sheet 385
Index 387

LIST OF PLATES.

PLATE.		FACING PAGE
I.	Fig. 1. Subchoroidal hæmorrhage from concussion and its relation to (*a*) "commotio retinæ"; (*b*) "holes at the macula"; (*c*) pigmentation of the retina; (*d*) pigmentary disturbance at the macula. (E. Treacher Collins.) (Page 112) . Fig. 2. (?) Colloid nodules in the choroid in a girl. (M. S. Mayou.) (Page 227).	114
II and III.	Figs. 1–4. An unusual case of opaque nerve-fibres. (Leslie Buchanan.) (Page 166) . . .	167, 168
IV.	Fig. 1. Papilloma of the conjunctiva. (M. L. Hepburn.) (Page 190) . . Figs. 2 and 3. Two cases of herpes zoster affecting the ciliary nerves. (M. S. Mayou.) (Page 226) . .	190
V and VI.	The sequelæ of hypotony. (E. Treacher Collins.) (Page 281) .	283, 287
VII.	Figs. 1–3. The sequelæ of hypotony. (E. Treacher Collins.) (Page 281) . Fig. 4. A case of detachment of the retina at the ora serrata. (Arnold Lawson.) (Page 231) . . .	291

LIST OF DIAGRAMS, ETC., IN TEXT.

FIG. PAGE

1—7. Colour-sense phenomena and some inferences which they seem to suggest. Being the Bowman Lecture. (Sir George A. Berry) 12—14, 20, 50, 52

8—10. Concussion injuries of the visual apparatus in warfare, of central origin. (S. A. Kinnier Wilson) . . . 100, 101

11. Small tortuous veins on the optic disc in association with small vascular nævi of the skin of the eyelid and of the nasal septum. (E. Treacher Collins) . . . 172

12—17. Protection of the eyes in warfare. (R. R. Cruise) . . . 177—182

18—25. Orbital suppuration. (M. S. Mayou) . 192—199

26. A further case of Leber's disease, and two allied cases, associated with changes in the sella turcica. (J. Herbert Fisher) . . 253

27. Two cases allied to Leber's disease, but without family tendency. (J. Herbert Fisher) . 259

28—32. The history of couching for cataract. (R. H. Elliot) . . 269, 270, 273, 274

REPORTS.

I. PRESIDENTIAL ADDRESS.

By E. Treacher Collins.

This is the thirty-seventh year of our Society's existence and the second time in the course of it in which we have had to mourn the death of a President. Those of you who were present at our last Annual Congress, will remember how Mr. Jessop's genial personality permeated the whole of our proceedings, and all of you will realise how deeply we have to deplore the loss of one who took such a keen and active interest in the progress and prosperity of our Society. Up to within a week of his death he was energetically engaged in promoting the success of the *British Journal of Ophthalmology* he had called into existence, and of the scheme for affiliating the ophthalmological societies throughout the British Empire, which will come before you for your consideration at the general meeting this afternoon.

Shortly after Mr. Jessop's death the Council of this Society met, and, on behalf of the Society, passed the following resolution, which I feel sure will meet with your approval :

> "The Council of the Ophthalmological Society of the United Kingdom records with deep sorrow their sense of the serious loss the Society has sustained through the untimely death of its President, Mr. Walter H. Jessop.

Throughout his long connection with the Society he was animated by the single purpose of promoting its best interests and of sustaining and enhancing its scientific reputation. None appreciated more than his colleagues on the Council the rich maturity of his professional attainments and the conspicuous ability with which he adorned the Presidential office.

His friendly relationship with the leading ophthalmologists of continental and foreign schools in no way diminished his ardent desire and his unremitting labours to further the cause of British ophthalmology, of which he was one of the most brilliant exponents.

The Council begs respectfully to tender to Mrs. Jessop their sympathy with her in her bereavement."

A copy of it was forwarded to Mrs. Jessop, from whom a graceful acknowledgment has been received in reply.

The Council then proceeded to consider how best the affairs of the Society could be carried on. They found that Rule 14, dealing with vacancies in officers, empowered them to fill up any vacancies which may arise between one annual meeting and the next. They decided to act in accordance with it and appoint a new President. It has been the custom for some years past to elect a President alternately from amongst members who reside in London and those who reside elsewhere. As, however, Mr. Jessop's tenure of office had not expired, it was thought best on this occasion to appoint another Londoner, and so it came about that you find me occupying the position in which I am to-day.

Mr. Jessop is a very difficult man to follow, but when I look at the list of names of former Presidents of this Society, I find that any one of them would, likewise, have been a very difficult man to follow. I can assure you to have to follow on the cumulative effect of all of them is indeed a most embarrassing procedure.

They have left behind them great traditions. Traditions which it will be my earnest endeavour to maintain, so that the Society may continue its course in a state of progressive efficiency.

We have during the past year had a further sad loss in the death of Mr R. W. Doyne; another dear friend of many of us; another brilliant British ophthalmologist, who was a frequent attendant at our meetings and an able contributor to our proceedings.

This is the third time we have held our Annual Conference whilst our country has been at war. If one of our successors wishes to look back and see what part his forerunners played at this important epoch in the world's history he will, I think, find it fairly well reflected in the volumes of our *Transactions*. The prevailing subject of our discussions this year, as in the two preceding ones, will be the injuries and maladies resulting from the war.

The longer the war goes on the more and more evident does it become how essential is the skilled experience and training of Opathalmic Surgeons in connection with it. Before the outbreak of war no provision had been made by the Army whereby it could avail itself of the services of Ophthalmic Surgeons. Now, several of them have received commissions in the R.A.M.C. to act as specialists in France, in the East, and in the Military Hospitals of this country. Numerous others, unofficially, and unrecognised, are engaged in the Voluntary Hospitals throughout the country, not only in attending to the eyes of the wounded, but also in rendering visual aid to those preparing for warfare, and those manufacturing its all-important munitions.

Before the sad event occurred which has placed me in my present position, I had prepared a paper to bring before you at this meeting; though, not dealing directly with a subject connected with the war, it is one to which my attention became directed from observations on its victims. It is a continuation of a paper I read to you last year on "Contusion Hypotony" and deals with the

"Sequelæ of Hypotony." I wish to bring it before you as a paper, and not as part of a Presidential Address, because I think it contains much debatable matter.

One after another we come before you at these meetings and describe the results of our observations, our experiments, and our reasonings. We are all, however, liable to illusions, misinterpretations, and unconscious bias. The best antidotes to these failings are criticism and debate; I hope you will apply these antidotes freely in the course of this Congress. Not criticism of an acrimonious or self-assertive character, but criticism having the same objective as that which is criticised, the elucidation of truth.

II. COLOUR-SENSE PHENOMENA AND SOME INFERENCES WHICH THEY SEEM TO SUGGEST.

BEING

THE BOWMAN LECTURE,

BY

Sir George A. Berry.

The privilege of being allowed to add to the series of addresses by which we celebrate the memory of our great first president, is one which one cannot fail to appreciate. The series has been so comprehensive and so brilliant that both the choice of a new subject and the desire to approximate to the standard attained by his predecessors must necessarily cause embarrassment to each successive lecturer.

When the Society did me the honour of appointing me "Bowman Lecturer" I suggested to our late president that I might take for my subject a discussion of the various wounds of the eye and other conditions affecting vision, caused by the war. This did not, however, satisfy the president, who indicated that something of more general scientific interest was expected. I have accordingly chosen a subject, which, while it may fall under that category, is at the same time necessarily highly speculative.

Those who have studied the present position of our knowledge of colour-vision will recognise that the subject is too extensive to deal with in a single lecture. They may admit too, that, notwithstanding the great accumula-

tion of facts of the highest importance and interest, which has resulted from the work of many able observers, little advance has been made in satisfactorily explaining the nature of the colour-sense.

My chief excuse, therefore, for taking up a subject, so difficult that it has baffled men of the highest intelligence and the most profound scientific attainments, must be, not that I can presume to contribute anything of value, but that any critical digest of the physiology or pathology of the colour-sense has not, so far, formed the theme of a "Bowman Lecture."

I can, however, lay claim to have interested myself in colour-vision, by experiment and clinical observation, during forty years. I do not, therefore, propose to offer only a *résumé* of the subject, but to indicate and give reasons for the standpoint to which observation and reflection have led me in regard to some of the points which call for elucidation.

As far as possible I shall discriminate between facts and hypotheses.

I.

We may, in the first place, consider the more or less evident, as well as the experimentally established, facts of normal colour-vision.

Firstly, it is evident that as far as physical stimuli go, colour is inseparable from light, whereas we may have light without colour. It is a daily experience that external objects present more or less colour in the daytime, or in sufficiently strong artificial light, and lose their colour when the surrounding light is reduced beyond a certain limit.

Again, we normally distinguish a very great number of colour-sensations which differ in their hue, in their brightness or luminosity, and in their purity or freedom from admixture with non-coloured light, *i. e.* in their saturation. All the hues, tints, and shades, which appear to us different when in juxtaposition with others, make up a very large series of colour impressions. Even the

recognisable hues alone, in their purest form, as evoked by stimulation with monochromatic light, are so numerous that normal colour-vision may be said to be polychromatic.

All these hues cannot receive separate names. Although there is a difference between the trained observer and others as regards memory for different hues, and, consequently, as regards the names which may be given to them, the greatest number of hue names could hardly amount to more than 5 per cent. to 6 per cent. of the hues, which, on juxtaposition, are separately recognisable. Therefore we commonly refer most of them to the two nearest hues for which we have a name. Thus, the colours from yellow to green would generally be referred to as yellow, greenish-yellow, yellowish-green, and green.

Another characteristic of normal colour-vision is that all the hues are felt to have a definite relation to each other. They can be placed, one after another, in a series which leads by gradation through the entire recognisable number. Thus, if we begin with a red we should place on one side of it a hue which appeared yellowish-red, and on the other side one which appeared bluish-red. Unless the differences were very slight we should arrange them thus without any hesitation.

Normal colour-vision is therefore not only polychromatic, it is characterised in addition by an inherent sense of hue relationship. When hues are very impure this sense of relationship is not so perfect.

The fact that we can only give such a small number of names to the separately recognisable hues causes us to group a number of different allied impressions under one name. Notwithstanding that we recognise more or less differences in hues of the same name, we are unable to say exactly to which single one of the hues so grouped the name is particularly applicable. Though we may limit the number of hues taken in each case we cannot pick out, as compared with the most closely allied colour impressions, what we without any uncertainty would say is *e. g.* a pure red or green or blue or yellow. That is to

say, we have no consciousness of a simple or fundamental sensation in any case. I refer to this as it seems to me to be a curious fact in view of the very widely-held belief in the existence of fundamental colour impressions. I am aware, of course, that these postulated fundamental impressions are held to be psychical ones which only approximate to, but do not wholly correspond to, any physically excited colour impressions.

Leaving aside, for the moment, the question of colour sensations other than those experienced in a good light and on central fixation, the next point to consider is that of colour mixtures. To some extent we have experience of this in ordinary life, but experiment is necessary for its better appreciation.

The laws which govern the mixing of pure monochromatic colours are :

(1) When two colours are mixed in equal quantity the effect is to give rise, either to an intermediate colour sensation, or to a neutral, non-coloured impression.

(2) If the quantity or the strength of the one colour is greater than the other the effect of mixing is to produce, either an intermediate hue which more nearly resembles the stronger than the weaker, or merely a weakened impression of the stronger colour.

(3) As the dissimilarity of the component colours of equal strength increases, up to the point at which a neutral impression is produced, the resultant colour becomes fainter and fainter and at the same time less saturated ; more overpowered by its admixture with non-coloured light.

(4) Mixtures made of colours near the two ends of the spectrum give rise to non-saturated hues which are not like any contained in the spectrum and are therefore colours which cannot

be elicited by stimulation from light of one particular wave-length only.

(5) The brightness or luminosity of the resultant colour is equal to the sum of the luminosities of the components.

(6) Similar effects are produced by mixing three, four, or more colours. When all the spectral hues are combined the effect is, of course, to reconstitute the white light from which they are derived, the luminosity of which is the sum of that of all the hues.

(7) To the larger proportion of the spectral hues there is another spectral hue which is its complement. If any two complementary hues be removed from the spectrum, the remaining hues, when combined, give rise to white. The same would be true however many pairs of complementary hues were withdrawn; the only effect of recombining those remaining would be a more or less noticeable diminution in the brightness of the resulting white light.

(8) The spectral hues which have no complements in the spectrum are complementary to hues got by mixing other monochromatic rays. Such complements are, of course, less saturated.

Saturated complements to all hues are got subjectively as after-images. There is no difference in this respect whether or not the hues have spectral complements.

The neutral or white effect, *i. e.* the luminosity of the different spectral hues, varies in degree. Some are more luminous than others. The most luminous part of the spectrum is not always the same, either in all individuals with normal colour-vision, or in the same individual in different states of the eye.

What has been said so far seems to justify certain inferences, which may be stated as follows:

(1) The polychromatic series of colour-hue sensations which characterises normal colour vision

is a re-entrant one. Starting with one hue, and arranging the hues according to their similarity, we can pass through the whole series and come back to the starting point.

(2) Half the hues in this closed series are complementary to the other half, so that hue and complementary hue, when combined, in each case cause a neutral or non-coloured sensation.

(3) As, in addition to its colour effect, each hue produces some neutral light effect, and as this neutral effect is always added to that of any other hue or hues with which a particular hue may be mixed, whatever be the resultant colour effect, the neutral effect must be looked upon as something apart from the colour effect. Complementary colours cannot, therefore, when combined, be held to produce or *make* white light. By mutually destroying each other they only *leave* a non-coloured sensation.

[*Note.*—We may represent the facts concerning the mixing of hues, as well as some of the inferences which may be deduced from them, by a simple geometrical construction.

Imagine all the separately recognisable hues arranged round a circle in the order of their relation to each other, complementary hues being placed opposite each other at either end of a diameter. Lines drawn from the centre of this circle may then be taken, by their direction, or the point at which they cut the circle, to represent different hues, and by their lengths the respective strengths of these hues. Two lines drawn from the centre in opposite directions would indicate the strengths of complementary hues. Any other two lines which do not deviate to the extent of 180° would therefore represent the positions and strengths of hues which lie between two complements, and which are more and more dissimilar the nearer their angle of deviation approaches to 180°.

We may (with certain reservations) take the two lines

to represent in magnitude and direction two forces, whose resultant, in magnitude and direction, is given by the parallelogram of forces. So far as the direction of the diagonal of the completed parallelogram goes, it will cut the circle at a point which gives the resultant hue. Experiment shows that this represents probably exactly what takes place. On the other hand, it would not be easy, if at all possible, to show how nearly the diagonal length also gives the colour strength, apart from luminosity, of the resultant hue compared with the sum of the colour strengths of the components. It can only be an inference that it does so. It would thus vary from a maximum, when the component hues were the same, to a minimum when one was the complement of the other, *i. e.* from the sum of the colour strengths of the components to their difference, or, if both were equal, from twice the strength of each to zero. So far as the extremes go, this also corresponds to what is found experimentally. But it hardly follows that the parallelogram of forces is altogether applicable.

It has to be remembered that both physiological and psychical factors must to some extent modify the completeness of an analogy based upon physical principles alone.

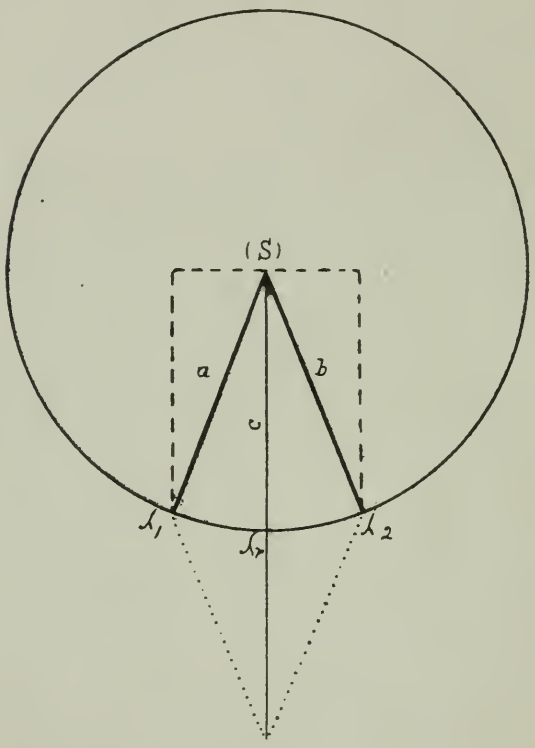
The difficulty of applying any experimental test of the accuracy of an inference of this kind lies greatly in the compound nature of the impression produced by the mixture colour. Its colour effect is modified by the degree of admixture of white light. This again depends upon the extent of dissimilarity in the two components as well as upon the specific proportion of non-coloured light which accompanies and is bound up with the sensations which they separately produce.

In the Figs. 1–3 the points at which the diagonal C (prolonged if necessary) cuts the circle, represents the position of the resultant hue λ_R. The length of the diagonal is the sum of the two components parallel to it. These go to form the new hue. The other two components at right-angles to C represent the degree to

which the two hues, λ_1 and λ_2 of colour strengths A and B, neutralise each other when mixed. These two pairs of components are shown by interrupted lines in the figures.

We have seen that the more widely the hues λ_1 and λ_2 are separated the more is the resultant hue λ_R found

Fig. 1.

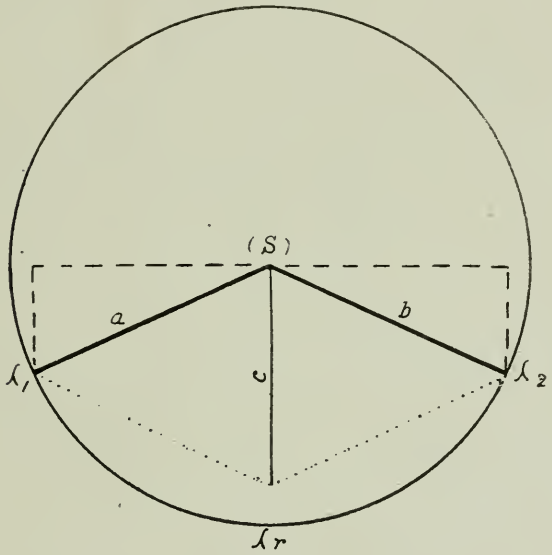


experimentally to lose in saturation. In accordance with inference (3) above, the components which make up S in the figures, while their colour effects are abolished, leave a non-coloured neutral effect. To this part, S, of the whole neutral effect associated with the two component hues I would attribute, hypothetically, an influence on the purity or saturation of the resultant impression.

It may be taken to represent the factor whose magnitude is inversely proportional to the degree of saturation of λ_R.

The luminosity of the resultant colour, on the other hand, cannot be shown in the same way because the resultant luminosity depends upon that of each component, and that is a quantity which varies with the hue. No doubt, from what is stated in law (5) above, the

FIG. 2.



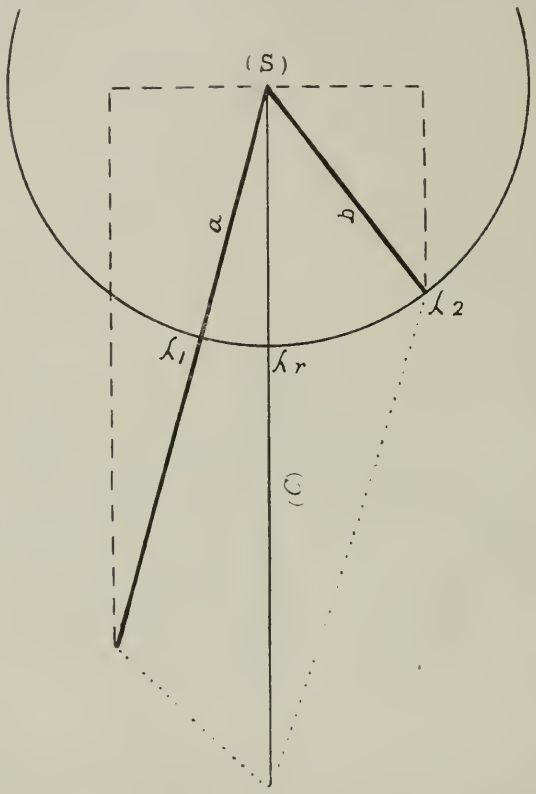
resultant luminosity is the sum of the component luminosities. On the above assumption, part of that summation of luminosities affects saturation.

The whole question of the relation of luminosity to saturation is obscure, and, therefore, this way of looking at it cannot claim to be anything but conjectural. It is consistent, however, with the relation which exists for monochromatic rays between luminosity and hue within wide limits of intensity. It is also consistent with the assumption, to which fuller reference is made later on,

that the neutral and hue effects of light stimulation are of independent causation though intimately associated.

The resultant colour impression, made up of hue, colour

FIG. 3.



strength, and saturation, but, apart from luminosity, would then in general be noted by C : S.

If we put θ for the angle between A and B, and α and β for the two divisions of that angle made by C, the relation,

$$\frac{\sin\ \alpha}{\sin\ \beta} \text{ or } \frac{\sin\ (\theta - \beta)}{\sin\ (\theta - \alpha)} = \frac{B}{A}$$

determines the position of λ_R.

For its strength we have—

$$C_{\lambda R} = (A^2_{\lambda 1} + B^2_{\lambda 2} + 2A_{\lambda 1}B_{\lambda 2} \cos \theta^{\frac{1}{2}}).$$

For A always > B and B = 1 this becomes—

$$C_{\lambda R} = (A^2_{\lambda 1} + 1_{\lambda 2} + 2\,A \cos \theta)^{\frac{1}{2}}$$

the maximum value for which is A + 1 when $\theta = 0°$, and the minimum value A − 1 for $\theta = 180°$.

As again by law (1) only equal strengths of λ_1 and λ_2 fully neutralise each other when these rays are complementary, the value of S is given by—

$$S = 2 \sin \frac{\theta}{2}$$

which has a maximum for $\theta = 180°$ and a minimum for $\theta = 0°$.

In general, therefore—

$$\frac{C}{S} = \frac{(A^2 + 1 + 2\,A \cos \theta)^{\frac{1}{2}}}{2 \sin \frac{\theta}{2}}$$

]

We may now consider the characteristics of normal peripheral colour-vision.

(1) To some extent they are the same, in the light-adapted eye, as for central colour-vision, with the exception that all colours appear somewhat fainter and less saturated, and the more so the more peripheral is the retinal image. Increase in the size or the brightness of a coloured object is required to produce the same impression as that received on central fixation, and the more so the more peripheral the image is.

(2) Within a zone intermediate between the centre and the extreme periphery, but nearer the latter, most colours appear changed in hue. This zone is not circular in shape, but varies in aperture (just as is the case with the field of vision for white, though not exactly in the same ratios for different meridians of the field).

(3) The result of this is that the binocular field of vision, for which the colour-sense is, qualitatively at least, comparable to the central colour-sense, is not co-extensive with, but larger than, the uniocular field (just as is the case, though again, not exactly in the same manner, as with the binocular, as compared with the uniocular, field for white).

(4) A narrow zone at the extreme periphery is practically devoid of all colour-sense.

(5) A white object, though it may appear less bright, never loses its neutral character, however excentric its retinal image may be.

The condition of matters in the intermediate zone calls for more detailed consideration. In it polychromatic vision becomes more or less suddenly changed to dichromatic vision. Only two definite hues, which are complementary—a particular blue and a particular yellow—appear in their natural hues, though less saturated than on central fixation. All other hues appear as more or less impure blue and yellow, *e. g.* bluish, mauve, yellowish, yellowish-brown, or quite neutral, *i. e.* grey. It is easy, *e. g.*, to select reds and greens of hues which, though all colour does not disappear from them in the dichromatic zone, yet produce similar impressions, of what one might call a more or less dirty or faint bluish or yellowish colour. It is also easy to select a red and green which both give an approximately neutral effect. Just as there is a couple of complementary hues which are unchanged in the impression they produce over the defective zone, so there is another complementary couple which do not cause any sensation of colour at all, but only a neutral impression.

It has to be remembered that it is not easy to feel sure, with a weakened colour-sense, in the case of excentric vision, whether all colour effect has gone or not. It is even difficult to tell a pure grey by direct fixation, though easier to do so when an object known to be sufficiently nearly grey is placed in juxtaposition to it.

For peripheral vision it is more difficult to make such a comparison. Altogether inferences in explanation of colour-vision derived from observations of peripheral impressions must be made with care and with some diffidence. I find that to make sure that a peripheral impression is one of pure grey, one has to test whether it gives rise to a coloured after-image or not. Even a colour which appears to be grey as seen at the periphery, when produced, *e. g.* by a light luminous green or pink, will give rise to a vivid, though transient, after-image projected against a white surface. This takes place, too, when the individual tested is not aware that the peripherally seen image is that of a coloured object. He will pronounce it grey, and yet will see the vivid flash of the complementary colour when the test object is withdrawn.

A less satisfactory way is to compare a grey to the inner side of the field of vision of one eye with a colour which becomes approximately grey when presented at the same time to the inner field of the other eye.

Therefore another characteristic of peripheral colour-vision is—

(6) That hues which give rise in the dichromatic zone to neutral sensations have no coloured after-images; that is to say, that there is a simultaneous inability to perceive in a normal manner both a particular hue and its complement. It does not follow, however, that the retina fails to react in a normal way to both or either. This point will be considered later, as it is a speculative one.

(7) One of the neutral hues of the dichromatic peripheral zone is not found in the spectrum; it belongs to that part of the closed colour curve which is not represented by monochromatic rays. This is a somewhat suggestive fact. In this respect, as in others, peripheral colour-vision, in the normally colour-perceiving, is similar to congenital colour-blindness of the usual type.

[*Note.*—A convenient way of studying peripheral impressions is the following: A tube, 10 cm. in diameter and 5 cm. or more long, is covered at one end by a screen of paper or thin cardboard, in which are punched two circular openings 25 mm. in diameter or less according to the size of the peripheral image required. The centres of the openings are separated by a distance of 60 mm.

An object, about 20 ft. distant, is looked at by bringing the other, open end of the tube as close to the face as possible, and turning the tube so that the line joining the centres of the two apertures is horizontal. We thus see two images of the apertures, coinciding and overlapping, through which the object fixed is seen, and two excentric apertures, through which rays pass to the inner fields of the right and left eye respectively. Objects, of which peripheral images are formed on one eye, are excluded from the sight of the other eye.

The after-images of peripheral impressions are studied by projecting them on white, grey, or black surfaces behind the test colours as these are quickly withdrawn.

This is the simple form of an arrangement which can easily be adapted so as to alter the size and illumination as well as the excentricity of either or both peripheral images, and thus admit of comparisons of the same or different colours simultaneously with both eyes or successively with one eye alone.

The variations in excentricity, visual angle subtended by the object seen excentrically and extent of area stimulated, involve the factors: The distance from eye to screen; (a) size of aperture in screen; and (d) inter-ocular distance. The last only is subject to individual difference, and need not, in view of the possible accuracy experimentally obtainable, be varied at all. It may be conveniently put at 60 mm. The two former are arbitrary variables.

The angle of excentricity of the centre of the peripherally seen image is $\tan^{-1}\frac{l}{a}$. The visual angle subtended by the

horizontal diameter of the aperture is sufficiently approximately $\tan^{-1}\frac{a}{l}\cdot\sin^{2}\tan^{-1}\frac{l}{d}$. The actual peripheral image is, of course, elliptical, with the long axis vertical, and this has to be taken into account in comparing areas stimulated.]

A rough analogy of a mechanical nature may represent some of the points so far referred to in connection with normal, central, and peripheral colour-vision. It is based on one first made by Krenchel about forty years ago, which appeared to me at the time to be suggestive, and to which I have often since referred.

Imagine a hemisphere floating in a liquid (Fig. 4). By force exerted in a line at right angles to the centre of its circular plane surface it can be pressed straight down into the liquid. Relief of the pressure would cause it to float straight up again. A force applied anywhere else but at the centre, and in any other but a vertical direction, would cause some other displacement. Leaving out of consideration any but forces acting at right angles to the plane surface, pressure at any point of the surface except its centre would cause a tilting. The hemisphere is free to tilt in any direction, and a force of definite magnitude acting at any point of the circular surface would cause a tilt in a direction which would be determined by the radius passing through the point at which pressure was made. The tilt would be greater or less according to the distance at which it acted from the centre. The effect of relieving the pressure would be not only a return to the stable position but a tilt in the opposite direction.

We may take the direct downward pressure as representing a sensation of neutral light, *i. e.* white or grey, and the recovery upwards as representing the after-image, which follows a neutral light impression. In the same way a tilt may represent a particular hue according to its direction, and the recovery the complementary hue.

We have further, in the freedom of tilt in all directions, an analogy with the polychromatism of normal colour-vision, as each hue may be represented by a tilt in the direction of a different meridian. The law of mixing of hues can also be represented by the same analogy, as

Fig. 4.

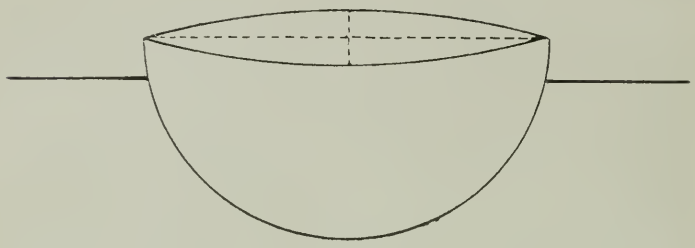
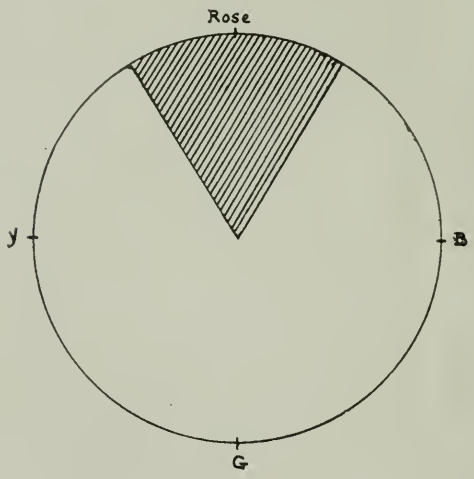


Fig. 5.



obviously the direction and amount of the tilt corresponds to the resultant of the direction and degree of pressure exerted in two or more meridians. An analogy to the summation of the neutral light effects would result on postulating that every force tending to tilt is accompanied by one which acts perpendicularly on the centre

and produces a direct downward pressure of the hemisphere.

We may postulate further, that a force cannot reach the hemisphere so as to be applied over a sector of its plane circular surface. Thus, in Fig. 5, the shaded sector might represent an area screened by a rigid surface. Any force acting perpendicularly to this surface may be assumed to be incapable of displacing the hemisphere either downwards or by tilting. The shaded area, therefore, represents the area over which no light vibrations can cause coloured or neutral impressions.

Again, it is clear that two equal forces acting along the same diameter at either side of, and at the same distance from, the centre, would neutralise each other. The tilt in one direction would be neutralised by that in the opposite direction. This would be analogous to the neutralising colour effects produced by two equally strong complementary hues.

The effect of binding down the hemisphere at two points of a diameter on either side of its centre, would be to reduce the possible directions of tilt from tilts in all directions to tilts in only two opposite directions. This, therefore, would be analogous to the change from polychromatic to dichromatic vision, the completeness of curtailment in the degree of freedom introduced in the case of the hypothetical hemisphere being also analogous to the completeness of the dichromatism, when, for two complementary hues, there was either a total absence of colour perceptibility, or only a more or less considerable defect in the impressions originated normally by stimuli corresponding to these hues.

We may carry the analogy a step further. Suppose that instead of binding down the hemisphere at two points on one particular diameter, we bind it down at three points not in line, it would then be deprived of any freedom of tilt. The same, of course, would happen if any more than three points not in line were taken. But obviously three points is the smallest number which would

have this effect. This, again, provides an analogy to the condition of achromatism.

Obviously, too, a selection of three points could be made to cause a tilt in any direction as well as to neutralise each other. There is, therefore, in this something analogous, not only to the neutral or white effect resulting from the simultaneous stimulation of three hues, but to the evoking of all other recognisable hues by varying combinations of three properly selected ones.

But the question naturally suggests itself: does it follow that because these three hues can on combination give rise to other hues, the three selected hues represent anything fundamentally different from any other recognisable hues? In other words, is there, on this account, any justification for designating normal colour-vision "trichromatic" instead of "polychromatic"?

There are, no doubt, other considerations for adopting such a nomenclature, but I believe the experimentally proved relation between trichromatism and polychromatism has no further significance than the analogous three points, at which forces varying in magnitude can cause every possible direction of tilt in a floating hemisphere.

"Trichromatic," as applied to colour-vision, is a term derived from hypothetical considerations. It represents what may possibly be the case, not what is actually known to be the case. On that account alone it is surely better to speak of polychromatic vision.

The floating hemisphere analogy may equally well be made to represent the two antagonistic pairs of fundamental hue sensations and the black-white pair of antagonists of the Hering hypothesis. But because it does so, this is no justification for inferring the existence of such fundamental antagonists. Indeed, in the absence again, of other experimental evidences in support of that hypothesis, the inference might rather be that such fundamental sensations had no actual existence.

We have now reviewed the main, more evident, or

most easily tested facts of colour-vision. There are, however, other facts which can only be arrived at by more or less difficult research, but about which something must be said before we can make any conjecture, necessarily speculative at best, as to the nature of the colour-sense.

Of these, we may now consider the following :

(1) Colour-brightness.
(2) Hue differentiation.
(3) Relation of neutral to colour-effect.
(4) Retinal light-adaptation.
(5) After-images and induction.

Colour-brightness.—An examination of the spectrum makes it evident that the hues seen in it are not all equally bright. It is found by different methods of investigation, which need not specially be referred to here.

(1) That the brightness, when the spectrum is got from a moderately strong light, is greatest for wave-lengths midway between the longest and the shortest which cause hue impressions, *i. e.* for a yellow.

(2) That for hue luminosity there is only one maximum, the increase being gradual from either end of the spectrum.

(3) That the position of maximum luminosity moves gradually more and more towards the shorter wave-length hues when the spectrum is derived from a considerably and increasingly less luminous source, until, for a very feeble light, it becomes fixed for a medium green hue.

(4) That for a strong illumination, the reds are appreciably brighter than the blues, whereas the opposite is the case for a feeble illumination. Thus, as illumination is reduced, reds become relatively darker and blues brighter.

(5) That the luminosity of the whole spectrum, when recombined as white light, is equal to the sum of the luminosities of the separate hues.

(6) That the effect of very strong illumination is to

alter the hue sensations in the direction of yellow and blue to begin with, and, finally, with the highest intensities, to make them all appear white.

Hue differentiation.—This varies for different regions of the spectrum. Unlike the luminosity curve, which shows only one maximum, to either side of which the fall is continuous, the colour differentiation curve is a more irregular one. Maxima occur in the yellow and blue, but there is no steady fall from these maxima to either end of the spectrum, where there can hardly be said to be any true hue differentiation possible.

Relation of neutral to colour effect.—This relation is one of the most interesting and important and cannot be lost sight of in any conjecture as to the nature of the colour-sense. A light stimulus must reach a certain intensity before a specific colour-sensation is elicited. This intensity varies in the case of the different spectral hues. Thus, in the dark-adapted eye the *minimum visibile* for neutral light or the minimal non-coloured perception which can be evoked, is the same whatever be the wave-length of the feeblest light stimulus. On the other hand, the *minimum visibile* for colour is elicited by a stimulus, the physical intensity of which has to be increased as one passes from the red to the violet end of the spectrum.

It may be noted that there is in this case something different from either brightness or hue discrimination, something which appears to be more intimately related to the physical basis of the specific sensations.

Retinal light-adaptation.—Adaptation for feeble stimuli, or what is called dark-adaptation, has, as it appears to me, mainly to do with orientation independent of colour sensation, *i. e.* it is concerned with the increase of sensitiveness to light, or the lowering of the *minimum visibile* for light, to admit of sufficient acuteness of vision as the surrounding illumination diminishes. Reference has already been made to the fact, and it seems one of the wonderful innate or acquired provisions of Nature, that in

the dark-adapted eye the greatest luminosity occurs for green, *i. e.* for hues which may be said to be the prevailing ones on the earth's surface.

It is in the dark-adapted eye that one can readily detect the colourless interval. But, although of profound importance physiologically, there does not seem to be any very definite connection between retinal adaptation and the colour-sense, which, after all, is more connected with light-adaptation. But some further consideration of this matter will be called for in connection with the more speculative portion of my lecture.

After-images and induction.—These phenomena are of the highest importance. They throw not a little light on the complicated nature of the colour-sense.

It is customary to distinguish between "successive" and "simultaneous" induction, which are closely related to each other. The former gives rise to after-images in the area stimulated and is not, I consider, properly speaking, an induction at all. The latter causes changes in surrounding areas, which are independent of or supplementary to their state of physical stimulation, and result from an interaction induced by conditions in the stimulated areas.

After-images occur both from coloured and from non-coloured light stimulation. And as these two always act together, when any sensation of colour is evoked, the after-image partakes of both the contrast to the coloured and to the non-coloured elements of the original stimulus.

The character of the after-image depends upon the nature of the light which has acted on the eye before any particular colour is fixed, upon the constitution of the colour fixed, upon the background on which it is seen, and upon the surface on which it is projected. The last condition does not, of course, apply if the eye is closed after the retina has been exposed for some seconds to the coloured object.

For central images, *i. e.* such as do not fall outside the fovea, the state of retinal adaptation does not apparently

have to be considered. Adaptation does, however, to some extent influence peripheral after-images just as it affects the vividness of the primary impressions.

If the after-image is got from a colour fixed in good daylight, the effect is that after steady fixation of a coloured surface for some seconds, the image, which the retina retains for a varying length of time with the eye closed, appears in the complementary colour to that which has evoked it. There is, therefore, a persistence, when the eye is closed, of the retinal image, but its colour is altered, becoming instead complementary to that of the objective stimulus. This complementary after-image is also darker in shade, if the colour fixed has been light, and lighter if the objective stimulus has been a dark colour. Black and white are, in fact, also complementary, so that the inherent white of some hues may lead to a dark after-image, while dark hues, or colours mixed with black, are correspondingly followed by a more luminous or lighter after-image. Further, if the coloured object fixed has an uncoloured background, that uncoloured surface also gives rise to an after-image. This after-image is not, however, one to which it would give rise were it not for the presence of the coloured object which it surrounds.

White, *e.g.* surrounding a colour seen under a visual angle of 10°, shows not only a dark non-coloured after-image, but one which is more or less tinged with the colour of the object fixed, and this tinge spreads out for a long distance. When the after-image of a green surface is projected on white its rose colour is lighter and the surrounding area appears as a greenish-grey. Projected against a black surface the rose colour is very dark and the surrounding area somewhat darker, and also, though less perceptibly, tinged with green. If the same size of green image is got without any background, by looking through a darkened tube which only takes in a 10° image, there is a considerable difference in the after-image. It is not so readily seen and is less vivid to the closed eye. Even when projected on a white surface it is

fainter, more decidedly grey, and the colouring of the surrounding area is not so apparent.

The colour of the object fixed causes, therefore, a simultaneous induction of its complementary colour in the area which surrounds the object, and the after-image of this induced complementary colour is its own complement, that is, the same colour as the object fixed. On withdrawal of the physical stimulus, therefore, the after-image of the central fixed coloured portion appears as the result of so-called *successive* induction. The surrounding white is succeeded by an after-image, which is the same, qualitatively, or nearly so, as would have resulted from an actual stimulation by light rays complementary to those producing the impression of the colour fixed. This latter is, therefore, an after-image to a colour *simultaneously* induced by that of the object fixed. In other words, it is a *successive* induction to a *simultaneous* induction, if the ordinary nomenclature is adopted.

While maintaining steady fixation of the coloured area, one is, as a rule, unconscious of the induced complementary tinge in the surrounding white. This is in striking contrast to the vividness which characterises the colour of the after-image.

II.

There are two links in the chain which ends in colour-perception about which our knowledge is fairly complete. These are the first and last links, viz. the physical nature of the stimulus and the psychical effect of that stimulus. We may assume, as most probable, that there are three intermediate links, viz. : (1) The end-organ or end-organs whose stimulation causes (2) the specific nerve conduction to (3) the visual cells or centre.

With regard to the end-organ link, it is obvious that, from the law of specific nerve conduction, there cannot be a very large number of different stimuli, each requiring its separate path of conduction along the optic nerve.

The number must at all events be negligible compared with the number of possible colour sensations. An elaboration of some kind must take place in the central nerve-cells. End-organs in the ordinary sense there must be, and these must, of course, be in the retina.

What, then, are the retinal end-organs for the colour-sense? Conjecture on this point suggests a consideration specially of:

(1) The anatomical structure of the retina.
(2) The neutral light effect of stimulation by monochromatic and combined rays.
(3) The colourless or, as it has also been called, the photochromatic interval.

Retina.—There are evidently, from what is known of the structure of the retina, three possible end-organs, all of which are different in structure and presumably also in their nerve fibre connections. These three are the hexagonal pigment-cells, the cones, and the rods.

As the pigment-cells, though showing some regional differences in size, are distributed over the whole retina, whereas the other two elements have a more circumscribed distribution, it seems natural to attribute to them primary importance in connection with that function of the retina which shows the least variation over the whole field of vision, viz. the light-sense. The position of those cells, in close proximity to the blood in the enormously vascular choroid, suggests that they are richly provided with some material which is acted upon by light, and which, as it is acted upon, is constantly replaced and restored to its photo-sensitive state. (Possibly, not unlikely, they contain more than one such material.) We know that pathological conditions which involve the retinal pigment layer, or impair its nutrition, are always associated with some light-sense defect. On the other hand, its want of nerve fibre connection shows that the changes which light produces in its cells cannot be directly transmitted, by nerve current, to the centres for vision.

The inference from this, taken along with the general

distribution of the light-sense, would be that photo-chemical changes in the pigment-cells served as an intermediate stimulus to both the other end-organs, the rods, and the cones. With regard to the latter, however, some difference in the manner in which this stimulus is transmitted by the optic nerve, or is received by the central cells, or both, might be supposed to exist, if the form-sense, which is so intimately connected with the light-sense, is, as seems generally to be believed, practically dependent upon differentiation of the impressions received by the cones alone.

The peripheral form-sense does not, however, correspond in acuteness at different points to the anatomical distribution of the cones. Can, then, the participation of the rods as form-sense end-organs be altogether excluded? It seems to be rather more likely that the less perfectly isolated and less numerous fibre connections of the rods, while subserving the purpose of the peripheral light-sense, may tend to make peripheral visual acuteness less than would be the case if it depended solely upon cone stimulation. In the dark-adapted eye, in fact, there seems some reason to infer that the rods are brought into action as less perfect form-sense end-organs.

The impressions to which rod stimulation gives rise provide also for a very acute sense of movement. This sense is dependent upon the light sense, though related to it otherwise than is the case with the sense of form, and is of great importance.

The macular cones, and possibly the peripheral cones as well, subserve also a subsidiary function. By their stimulation a nerve current passes to the cells, whose fibres innervate the sphincter pupillæ. This affects the size of the pupil in correspondence with the actual, and the temporarily relative, degree, of their stimulation. It seems not impossible, though there is no evidence in support of such an assumption, that there may be a somewhat similar relation between the stimulation of the rods and the reflexes leading to ocular movements,

and, therefore, indirectly to peripheral projection. The external projection of peripheral images may be associated with the sense of innervation of the oculomotor muscles.

Whatever may be the exact relation between the functional activities of rods and cones as form-sense end-organs, there can be little doubt that the cones alone represent the end-organ of the colour-sense. It is most likely, too, that just as for the form-sense, the stimulation of the cones which results in colour is an indirect one from changes produced in the hexagonal cells. Possibly they are directly stimulated by light.

Neutral light effect.—All rays, besides their specific colour effect, cause a neutral, non-coloured effect. In the case of monochromatic rays, except at either extreme of intensity, the two effects are inseparably associated. Whereas, however, the combined action of two or more monochromatic rays gives rise to a qualitative change in the colour effect, which may even result in all colour effect being abolished, there is no antagonism in regard to the non-coloured impressions which they produce. These effects are all summated in a physiological sense, *i. e.* they supplement each other.

In this there is such a radical difference that the two effects, though associated, can hardly be imagined to arise from the same kind of end-organ stimulation. Helmholz, referring to the neutral effect produced by the simultaneous stimulation of three colours, says: "As luminosity cannot be a linear function of the colour values it must be a perception which results from a more complicated combined action of the three elementary colour impressions." Instead of assuming that any, however intricate, combination of two, three, or more so-called "elementary" impressions can account for the association with colour of non-coloured effects (an association, no doubt, on which their luminosity depends) it seems more in accordance with actual facts to look upon luminosity as originating independent of colour and only so far associated with it, in that the same stimulus neces-

sarily, as a rule, *i. e.* when sufficiently strong, gives rise to coloured and non-coloured sensations which cannot be dissociated in their psychical effects.

The photo-chromatic interval.—In this respect there is, as I have already said, something which seems to connect the physical stimulus with the psychical effect. That is to say, there is a continuous change, according as the rays are of smaller and smaller wave length, in the proportionate stimulation which is called for to elicit their two associated effects—the non-coloured and the coloured.

Although the difference in the behaviour of the neutral and coloured effects in the case of compound light would lead one to infer that there was a different channel for their transmission, this alone would hardly be sufficient to account for qualitative differences in the one of these effects. On the other hand, it does seem possible to suppose that proportional or character differences in the two responses, aroused by the same physical stimulus, might lead to qualitatively different impressions.

We have, then, shortly to look upon the hexagonal cells as the intermediate between light vibrations and the rods and cones. We have to suppose further, that stimulation may be single in the case of the rods and two-fold in that of the cones. Apart from minor interferences with the perfection of the colour-sense, such as may be caused by the yellow pigmentation of the macula and by chromatic aberration, the eye itself is probably not otherwise associated with qualitative and other specific characteristics of the colour-sense (such as complementary after-images) than as providing the end-organ for transforming light vibrations into two different nerve currents which stimulate the central cells. Or it may be that the two different end-organ stimulations impose upon the conducted nerve current some periodic or other change which leads to a double response in these cells.

It is not impossible, too, to find on this conjecture an explanation for the fact that very intense stimulation causes a change in hue. Any change in the proportional

response to the double stimulation might be expected to alter the specific character of the final impressions.

So far as cone stimulation goes, I have assumed that that there are two elements of a cone simultaneously acted upon by light. In some way a consciousness of the proportional effects produced by the current or currents set up by these excitations in the central visual cells gives rise, it is suggested, to colour-perception. Supposing this to be the case, the same, or at least approximately the same, proportion must be maintained through a wide range of illumination. Therefore, the curves of excitability of the two cone end-organs must be presumed to be similar over a corresponding range. Further, the responses in the central visual nerve-cells must be the same relatively through the same range.

I have assumed also that one of the conducted currents, or possibly one element of a more complex current, gives rise to a central disturbance which causes a non-coloured light sensation alone, *i. e.* the perception of luminosity or whiteness. The more intense this current the greater will be the luminosity ; and, as long as the responses to the two currents keep pace with each other, or maintain the same proportion to each other, the greater the intensity of the double stimulus, the greater will be the brightness of the particular unaltered hue. Now, in accordance with physiological facts, we have only to suppose that, beyond a definite limit of intensity, the normal relation between the two impressions is disturbed. The brightness or whiteness continues to increase, but the changed relation causes the hue to alter, to lose more and more in saturation, and eventually to disappear. Whether this can be ascribed to a failure in central response to the element of the current which has no neutral effect, or to a difference in excitability of the end-organs, is a matter as to which no data exist for determining. From considerations, which will be referred to later, the former would appear to be more likely.

Certain observed facts, in connection with the photo-

chromatic interval, call for consideration, as they might appear to be inconsistent with the above assumption of a constant relation, differing for each hue, between the two postulated stimuli upon which their qualitatively different perception-evoking effect depends. Thus, the colour interval is only readily demonstrable for peripheral vision; there is even some doubt as to whether it exists at all for foveal vision; further, the interval increases, for peripheral vision, with dark-adaptation. In this case, therefore, the ratio between neutral and colour stimulation alters. On the other hand, there is no qualitative, but only a quantitive, colour-sense difference for the periphery of the field of vision until we come to the dichromatic zone. Therefore, either the assumption of a constant relation being maintained within wide limits is fallacious, or there is some other explanation for the difference between the foveal and the extra-foveal retinal areas in respect to the colour interval phenomenon. This, I believe, is to be found in the complication with rod stimulation. It may well be, indeed, there is some reason to suppose it most probable, that peripheral *cone* stimulation does not differ essentially from foveal cone stimulation. The fact that rod stimulation becomes possible for enormously diminished intensities by dark-adaptation, so that the non-coloured impression threshold is thereby more and more lowered, does not entail the assumption that the peripheral cones as well, are, under similar conditions, either more readily stimulated or altered in excitability. Like the foveal cones they are probably to a great extent unaffected by adaptation, at all events in the same sense as the rods.

This relative behaviour of rods and cones is indeed one of the reasons for assuming that only the cones have to do with colour vision. It also points to some participation of the rods in the peripheral form-sense to which reference has been made.

While, therefore, the facts just referred to do not appear to negative the view I have suggested, it may be

admitted, on the other hand, that they do not afford any evidence in support of it. Nevertheless, it is the character of the photo-chromatic interval, as revealed on dark-adaptation for extra-foveal vision, considered in relation to its dissimilarity to the spectral luminosity curve, which suggests a double-cone stimulation. Possibly only the characters of the two stimuli may be different and vary in that respect for different hues. There are experimental difficulties in demonstrating any colour interval for foveal vision, and although it seems most probable that such an interval does exist, it is not an essential condition for assuming the existence of a double-cone stimulation.

The peripheral dark-adapted photo-chromatic interval phenomenon is complicated also by the altered luminosity values which adaptation involves, but this does not bear upon qualitative colour perception.

Nerve conduction.—The correct perception of colours implies a normal state of conductivity in the optic nerve fibres and their end-organ connections.

A study of the physiological conditions which influence the delicacy of the light, form, and colour senses enables one to understand how alterations in conductivity explain the imperfections which are associated with diseased conditions. We have only to consider the physiological conditions under which the strength of excitation of individual retinal end-organs is altered, and the effect of this on the visual functions, to account for the same effects where a diminished excitability, or a diminished conductivity, results from pathological changes.

For the normal eye the visibility of any object is determined by three factors:

(1) Its luminosity.
(2) Its contrast with the background.
(3) The visual angle which it subtends.

In order that two points may be seen as such, *i. e.* separated, and not merged into one, there is a limit beyond which the visual angle cannot be reduced. This

limit, in an emmetropic eye, is dependent upon, and therefore varies with, the brightness of the two points and the contrast between them and the intervening space. Up to a certain degree of intensity the limit is lowered, and consequently the visual acuteness increased, by increasing the brightness of the two points. As to contrast: in the case of minimal visual angles this has to be much greater to admit of separate distinction than when the objects and intervening space subtend larger angles. That is to say, the light-difference sense is more acute for large than for minimal visual angles. Then, again, an object can be so little luminous that, if it subtends a very small visual angle, it is altogether invisible, whereas one as feebly luminous, if it subtends a larger angle, is seen without difficulty. This is explained by the physiological fact that the simultaneous excitation of a number of contiguous end-organs evokes impressions from very much smaller stimuli than are required in the case of the excitation of only a few end-organs. There takes place a summation of stimuli which carries the resultant effect over the threshold of sensibility. From this we may infer that a diminished excitability caused by disease may, within certain limits, be too small to assert itself where even a number of end-organs are simultaneously stimulated.

Anything, therefore, which diminishes the impression made by stimulation of a minimal number of retinal end-organs has physiologically the effect of reducing the acuteness of vision. When pathologically the impression, caused by objects seen under a very small angle, is diminished, as the result of defective nerve conduction or reduced excitability, the result must be the same.

Consequently, in neuritis, although there may be no great reduction in acuteness for ordinary black type on a white background, we find a more or less marked lowering of acuteness for grey on grey types, *i. e.* for objects contrasting less strongly with their backgrounds.

Again, in the relative central, or paracentral, scotoma of

tobacco amblyopia, the effect of the same light-sense defect for minimal spatial stimulation is observable in the disappearance of a small white object (which is easily seen outside the scotoma) when its image falls within the scotomatous area. On the other hand, the summation of contiguous impressions causes the appearance of a large white object to differ little, if at all, whether its image falls without or within the area of the scotoma.

The influence of the same elementary defect of the light-sense in this affection is seen in the reduction of the colour-sense over the area of the scotoma. The relative scotoma is therefore usually, though not so correctly, referred to as a colour scotoma. This comes from its being more readily demonstrable with coloured test objects.

Defective conduction, which causes the rise in the *minimum visibile* for non-coloured light, is accompanied by a corresponding rise in that for colour. The relative scotoma, therefore, shows a degree of colour-blindness demonstrable for coloured objects which are seen under a small visual angle, or which present a low degree of saturation. At the same time larger and more saturated coloured images are recognisable and either cause a normal impression, or one which is approximately normal, according to the density of the scotoma. This, again, is the same phenomenon of summation.

It is natural, therefore, that diminished conductivity in the optic nerve should lead to some colour defect. A defect from this cause is, however, always associated with a light-sense and form-sense defect. In this respect it differs from normal peripheral, and from congenital, colour-blindness. It differs also in other respects, as was clearly shown by the Royal Society Colour-vision Committee of 1890–91. Although the plate published by that Committee, p. 4 of the Report, undoubtedly does not give a correct representation of the congenital colour-blind spectrum, that which indicates the defective colour sensations induced by disease is probably sufficiently accurate for some definite degree of pathologically lowered

optic nerve conductivity. The former was based entirely on theoretical considerations, and did not even satisfy the only colour-blind member of the Committee; the latter is the result of a comparison, made by individuals, of their altered, with their normal, colour sensations.

If the explanation which I have suggested, that colour sensations arise from a central elaboration of the relation between two kinds of stimulations of the cones, represents anything approximately true, it does not seem too improbable to assume that the altered state of nerve conductivity may at the same time cause a disturbance in their normal relation. I have suggested that such a disturbance, though not necessarily of the same nature, would explain the altered hue sensations arising from extremes in the intensity of stimulation in the case of the normally colour-perceiving eye. Be this as it may, and notwithstanding that it is not easy to explain the relatively less imperfect transmission of blue in pathological cases, the suggestive fact is that colour defects induced by abnormal conditions of the optic nerve, in addition to being always associated with light and form-sense changes, are different from either physiological or congenital colour-blindness.

The visual centre.—In the inferences to which the known facts of colour-vision have led me so far, the *rôle* of the retina has been taken to be limited to the setting up of two different nerve currents, or possibly to only one current whose character is the result of two different stimuli. This has been assumed to be due to the physical stimulus of light acting indirectly on the cones through chemical changes which the ether vibrations produce in the substance or substances secreted in the hexagonal cells. The two end-organs in the cones, or the two chemical substances in the pigment-cells, I have assumed to be related in different ways in their response to the same light stimulus. The result of this, it is suggested, is that the single current evoked is different, or that the two currents bear a different proportion to each other, for

different wave-lengths in the ether disturbance. Within certain limits this proportion is assumed to be constant, and to be in some way the instigator of qualitatively different impressions in the central cells to which the currents pass. It is further assumed that conduction does not cause any disturbing effect on the proportionate stimulation when it takes place normally and under usual conditions, but does so when pathological changes exist in the fibres of the optic nerve.

All this is no doubt speculative enough, but, as I have attempted to show, there are physiological facts which may be adduced in the support of some such mechanism. When we come to the central cells an explanation of their behaviour must necessarily be even more purely speculative. I have pointed out that whatever be the nature of the retinal response it must be assumed that a central elaboration takes place before we arrive at the completed qualitative sensation of colour which characterises the psychical end of the process.

Of the psychical processes themselves we cannot offer any explanation. But there are certain facts, to some of which I have drawn attention, that point more or less clearly to the setting up, by the specific stimulation of the retinal end-organs, of some kind of molecular vibration or other change in the central cells.

In this connection we may consider :

(1) The re-entrant character of the relation of the various hues to each other.
(2) The effect of mixing hues.
(3) The sensation of black.
(4) The phenomena of induction.

The analogy of a floating hemisphere was introduced to illustrate some facts of colour-vision apart from the form-sense. It was not intended to indicate in that way the nature of any actual condition of any part of the mechanism of vision generally, or of colour-vision in particular. But some molecular movements, *e. g.* linkage changes, must be supposed to take place in the cerebral

cells. Whatever these are they must be subject to similar conditions as those which, by analogy with colour sensations, were seen to apply to the hemisphere. That is to say, they must be of a nature which may graphically be indicated by what, in a geometrical sense, one would call "direction." We may assume that the "direction" of the particular strain or other disturbance determines the qualitative colour sensation, while the amplitude of the disturbance is the measure of its intensity. How these molecular movements should evoke colour sensations is a psychical problem which hardly admits of any conceivable solution.

There are no facts either to indicate whether the different molecular disturbances all occur in the same cells, or whether those evoking colour perception take place in cells which are separated from, but closely related to, those by which non-coloured perception is evoked. But that the cells, if there are two classes of them, must be intimately interrelated, not only functionally but also spatially, seems to be indicated by the absence of any pathological evidence of the existence of a special colour-sense centre.

Let us suppose, then, that strains take place in all "directions" in a medium which, within certain limits, is perfectly elastic, and we have at once something similar to the freedom of tipping movement in all directions of a floating hemisphere. Or to take another and perhaps more complete analogy, we may compare equal elasticity in all directions with the molecular state of a crystal which freely transmits vibrations of ordinary non-polarised light.

The increasing weakness of a colour impression which takes place on the prolongation of a stimulus of constant intensity may possibly be taken to indicate, either that the central strain cannot be kept up indefinitely with the same energy, or that the elastic resistance to the force increases with the duration of the strain. The supposed reaction of the central cells might, therefore, possibly be looked upon more as a vital resistance than as elasticity

in the physical sense. On the other hand, the phenomenon may be more directly connected with the effect of prolonged action of light on the strength of the electrical stimulation produced by chemical changes in the retinal pigment-cells, *i. e.* it may have a peripheral, not a central cause. In any case the cessation of the stimulus causes a recovery from the strain in the central cells leading to an oppositely directed disturbance in them, and consequently to the perception of the complementary colour. This swing back in the opposite direction is not, however, as a rule, sudden. Yet a momentary stimulation may be supposed to cause an oscillation back and forwards. This would account for the observed fact that if such stimulation is sufficiently intense, a short, alternating series of positive and negative after-images results, which successively diminish in intensity till they cease to appear. The phenomenon suggests, in fact, an analogy with the trembling of a jelly which has received a sudden blow.

The normal condition of the cells connected with the macular end-organs may be taken to be one in which the resistance to strains or vibrations is equal in all "directions." The completeness of the closed series of colour sensations, if we include those which can be subjectively elicited, does not suggest any impossibility of evoking disturbances in some definite directions such as one had to impose upon the figurative hemisphere, to represent by analogy the fact that some hues are altogether absent from the spectrum.

It does not seem to be too great an extension of the supposition I have made to suggest further that all cone-connected cerebral cells are not alike, and that there may be some of them in which elasticity is more or less limited in certain directions. The effect of a complete loss of elasticity in one direction would be analogous to the condition of a crystal which caused plane polarisation of light. The effect on colour perception would be a change from polychromatism to dichromatism. This might be taken to be the condition of the cells which receive their

stimulation from the cones in the peripheral area of the retina corresponding to that of the field for which there is a physiological dichromatism.

Whether or not there might be assumed to be a gradual diminution of elasticity, culminating, for the central cells connected with this area, in complete loss in one direction, is a point which the known facts hardly afford grounds for surmising. Certainly the stimulus required to arouse colour perception has to be higher the further we pass to the periphery. But this is perhaps more likely to be due to the fact that equal areas of the retina contain fewer and fewer cones the more peripheral they are.

Then there is the analogy with congenital dichromatism, which, when complete, may be supposed to be due to the same rigidity in one direction of *all* cone-connected central cells. There are undoubtedly congenital cases in which there is only partial dichromatism. In regard to these, the same reasoning would lead one to infer the possibility of diminished, and not altogether absent, elasticity in the same cells. If, therefore, one held the view that complete congenital dichromatism resulted from the same condition of the central cells as that which normally characterised those connected with a peripheral zone of the retina, one would ascribe congenital incomplete dichromatism to the existence, in these cells, of a less complete abnormality of the same nature.

Again, there is the complete achromatism of the extreme periphery of the field of vision. As the cause of this we must presume that the central cells, which connect with the most peripheral cones, are in a state of more complete rigidity, *i. e.* that the molecular disturbances which elicit the sensation of colour, apart from light, do not follow at all in response to normal stimuli. There seems, anatomically at all events, no reason to ascribe the total colour-blindness of the extreme periphery to absence of cones.

We should, then, on the assumption which has been made, have at least three conditions of the cone-connected

central cells. (1) One in which molecular disturbances are possible in all "directions," and in which there is unrestricted elasticity. (2) One in which disturbances in one "direction" cannot take place at all, and (3) one in which no specific colour disturbances are possible. It is, of course, also conceivable that there are cells for which intermediate conditions may exist.

Whereas the existence of the second of these postulated groups would afford an explanation of both peripheral and congenital dichromatism, the third group cannot be inferred to offer an explanation of congenital achromatism as it is generally met with. These cases are rare. I have not personally had an opportunity of observing the condition. There seems, as a rule, if not always, to be more or less defect of central visual acuteness and nystagmus. In addition to this the luminosity curve for different monochromatic rays is different from that which characterises the achromatic periphery of the normal field. The inference is that congenital achromatism has a peripheral and not a central causation, and is due to some pathological condition of the retinal cones, which may also be more pronounced in some cases than in others. The possibility of cases existing in which the symptoms are less conclusive of a peripheral origin, more analogous to those of physiological peripheral achromatism, and therefore probably of central original, need not perhaps be altogether excluded.

It seems reasonable to suppose that the process of development in nature has been from group (3) to group (1). Occasional reversion to the earliest type is, therefore, conceivable. The comparative physiology of colour-vision, so far as inferences can be drawn from the results of necessarily somewhat inconclusive experiments, indicates the existence of states of colour-vision in animals which may, at least roughly, be referred to two of these categories—polychromatism and achromatism.

Congenital dichromatism, which is so similar to peripheral normal dichromatism that it suggests a common

cause for both conditions, may, therefore, possibly also be regarded as a reversion to an earlier type of response in the central cells to physical colour stimuli.

The differences exhibited by different cases of congenital dichromatism may be assumed to be partly due to differences in the "direction" in which the molecular disturbances are incapable of taking place in the central cells and partly to incompleteness of the elasticity abnormality, which may cause impaired molecular mobility. Though the so-called red-blindness and green-blindness cases are no doubt sometimes sufficiently distinct to warrant their clinical classification into different groups, they need not be considered to have such essentially different origins as is inferred on the Young-Helmholz hypothesis.

The sensation of black.—As all light stimuli produce an impression of luminosity and colour it follows that the absence of any impression of that kind must at least indicate the absence of any specific objective stimulation. Yet the sensation of black is just as definite a sensation as that of white, or of any colour. It is a sensation of quite a different kind that we experience in connection with external objects situated anywhere beyond the limits of the field of vision, of anything at the back of our heads for instance.

In some respects black may be placed alongside of the colour impressions which cannot be elicited by monochromatic light. There is, of course, this difference that these colour impressions can be evoked by physical stimuli. They may result from mixing two or more spectral hues. But the vivid saturation of these colours when they arise subjectively as after-images to certain spectral hues, is strikingly greater than can be got by any mixture of the physical stimuli. Indeed, this applies also in the case of all spectral colours. Under certain conditions a more saturated subjective colour can be got for all hues. This is the case when the after-image of any colour is pro-

jected on to its complementary colour. It is obviously due to the greater amplitude of the same molecular disturbance which would evoke the after-image colour.

In my own experiments with "black" I have used what might, from a physical point of view, be called "absolute black." This is got by cutting a hole in a deep box lined inside with a rough surface coated with lamp black. Yet the sensation to which this gives rise is by no means as black a sensation as can be got subjectively. For, if a white surface be held for a few seconds in front of a part of the hole, the area which it covered, is found, when it is withdrawn, to be very appreciably blacker than the rest of the hole. But also, in respect to its after-image, this "blackest black" acts to a certain extent like white or colours. If surrounded by a colour the black gives an after-image which is made up of that colour, or approximately that colour, mixed with grey.

We have, therefore, in a case for which objective stimuli can be excluded, conditions which are similar to those which may, and do, result from a specific stimulus. And, as in the one case, we have assumed a molecular disturbance in the central cells, the "direction" or character of which is opposed for colours which are mutually complementary, we may also infer that the black-white series of impressions arises from similar oppositely directed, or otherwise directly opposed, disturbances. There is, however, this essential difference that the black-white series never neutralise each other. While opposed in direction they are not antagonistic, but act like two non-complementary colours, so as to give rise, on combination, to an intermediate impression—grey.

Now, we must look upon the absence of colour, where there is any sufficiently strong light stimulus at all, not as a passive condition, but as caused by an active central molecular disturbance in some "directions," counteracted by actively oppositely directed disturbances. This results in equilibrium for the time. The passive state can only exist in the absence of all stimuli. In the case of the

black-white series of impressions disturbance in one direction would, on this assumption, correspond to a movement from grey to black, and in the other direction from grey to white. The preponderance in either one or other direction would determine the shade of grey. There are many instances of equilibrium established by active forces in opposition. The medium size of the pupil in daylight, *e. g.*, results from an active innervation to contraction counteracted by an active innervation to dilation.

It must be remembered, too, that the conception "white" is a relative one. Our ultimate sensations are, in fact, relative not only as regards non-coloured impressions but also with respect to the whole series of colour impressions. What appears white in daylight continues to produce the white sensation, even when under artificial illumination, its true character may really be greatly altered.

What does change, however, according to the nature of the surrounding illumination, is the effect of colour impressions, though even this is not necessarily appreciated. The knowledge of the colour in daylight may influence one's judgment. The differences come out most markedly when the after-images of coloured objects, as seen in daylight, are compared with the after-images of the same objects seen under some artificial lights.

The point which has to be emphasised is that the position of the sensation "black," which is evidently a subjective sensation, indicates by its practically complete analogy to colour sensations, that the site of both is a central one.

After-images and induction.—As to after-images I have given reasons for inferring that they result from a reversal, on the cessation of an active stimulus, of the character of the disturbance which that stimulus has set up in the cells of the visual centre.

There are, however, two facts connected with this subjective phenomenon which call for further consideration. To one of these incidental reference has been made, viz.

that the after-image is not always complementary to the colour impression evoked by the objective stimulus. Often it is far from being so. The reason of this, I believe, is to be found in the misconception which may readily arise as to the true nature of the exciting stimulus. It has to be remembered that, under different illuminations, and owing sometimes to light transmitted through the sclera, as well as under different conditions of stimulation affecting contiguous areas, the disturbance in central cells corresponding to a local circumscribed stimulus may be modified in a manner which is not consciously appreciated. In other words, the psychical effect of the central cell stimulation is subject to some variation. The judgment, therefore, is at fault. In this respect it is immaterial whether the local stimulus is one which ordinarily evokes a neutral, or some definite colour, sensation. We may continue to regard a white object as white even when, owing to changes which take place in the central cells, from induction, its non-coloured character should have disappeared, and, if our judgment were correct, a correspondingly altered impression should have resulted.

The fact that the colour of the after-image may be quite pronounced, although there may be no consciousness of the change in the white surface itself during its fixation, has already been referred to. Earlier in this lecture I pointed out the very striking vividness of the coloured after-image to an apparent grey as seen at the periphery of the field in the neighbourhood of its dichromatic zone.

The other fact in regard to after-images which calls for some notice is their recurrence. They fade away and then recur, often a good many times before they altogether cease to be observed. This phenomenon is most noticeable if the eyes are closed after the object has been fixed for some time, *i. e.* in the dark, and least noticeable if the after-image is projected on to a white surface.

If, *e. g.*, one fixes for thirty seconds a red disc, subtending an angle of 10° on a white surface, there is at first a very vivid after-image of the disc, in approximately

its complementary colour, the exact hue being dependent upon the purity of the surrounding white. This fades away and in a few seconds is succeeded by another image of the disc of a colour which is not quite the same as the first. Successive green recurrences take place, in which the green always appears less and less saturated, until the disc, in one of the recurrences, appears to be almost pure grey. After this there may be still more recurrences, always fainter and fainter, but becoming more and more tinged with red, until they finally cease to appear.

At the same time the after-image of the area surrounding the disc undergoes changes with each recurrence. At first it is distinctly red and not very dark. It soon, in subsequent recurrences, becomes much darker, though retaining this red tinge, until the after-image of the disc has passed through grey to become tinged with red, when the surrounding area becomes lighter and at the same time greenish.

This one example illustrates the phases which occur, *mutatis mutandis*, when other colours and surroundings are taken.

The fact that the after-image of any colour, if projected on its complementary colour, heightens the saturation of the impression which that complement produces, indicates that an after-image is caused by a molecular disturbance of the same nature as, but opposite in direction from, that which the physical stimulus originates.

The nature of the recurrences of the after-images shows in addition that this complementary colour-originating disturbance slowly diminishes in amplitude until the sensation which it causes has sunk below the threshold of colour perception. It shows, too, that the disturbance then regains the character which was set up by the original stimulus, causing thus positive after-images.

The fading and reappearance of the after-image takes place in the absence of other sufficiently strong stimuli over the same area of the field, such as occur when it is projected on a white surface. To get the full sensitive-

ness to the fainter central disturbances the eye must be at rest.

In addition to recurrence there may be, under certain conditions, a change in the actual colour of the after-image on successive reappearances. This must also be due to induction. Or, rather, it seems to be due to a difference in the proportionate strengths of the physically elicited and the induced disturbances. The latter, presumably on account of smaller amplitude, produce an increasingly weaker effect on each recurrence. Thus, a red which alone, *i. e.* when surrounding objects are cut off, is followed by a very definite green after-image, gives, when surrounded by blue, a very distinctly blue after-image. But, on each successive recurrence its own green after-image asserts itself more and more strongly, even although the colour impression becomes gradually more and more faint, as the amplitude of the disturbance diminishes.

The interaction which takes place, presumably in the central cells, goes by the name of *induction*. Whatever be its nature it is at least evident that the process induced in the cells in the neighbourhood of those which respond to a definite objective stimulus, only differs from that in the directly stimulated cells by being oppositely directed.

This induction takes place, too, practically instantaneously, and also equably, over a large area. Consequently, the after-image phenomena are the same whether they follow a physical (objective) or an induced (subjective) stimulus.

With regard to uncoloured surfaces: the colour hue of their after-images depends upon that of their surrounding. This is modified, however, by their own non-coloured after-images. There is always a combination of an after-image to the physical stimulus in the areas occupied by the non-coloured images and an after-image to the colour which is subjectively induced in the centres corresponding to the same areas dependent upon the colour stimulation of surrounding areas.

White, grey, or black surrounded by red appear in after-image as dark red, red and light or pinkish red on a greenish or bluish-green background.

If neutral, non-coloured, surfaces are interposed between two different colours, the after-images of the neutral surfaces have approximately the colour which would result by mixing the two background colours modified only by their own non-coloured after-images. The resultant impression is the same as if both stimuli were objective instead of the one being subjective, *i. e.* induced. The inductive interaction takes place in all directions, and is therefore both centripetal and centrifugal.

The reason why the neutral surface after-images are not shades of, exactly, but only approximately, the resultant hue of the surrounding colours, is no doubt that the same process of induction which causes a colour effect in the areas stimulated by non-coloured light also leads to an interaction between the two colour-stimulated areas.

What takes place may be illustrated by a figure (Fig. 6) in which the two colours are denoted by A and B and the neutral zone by N. The respective after-images in the absence of a disturbing effect of induction may be denoted by A', B', and N'.

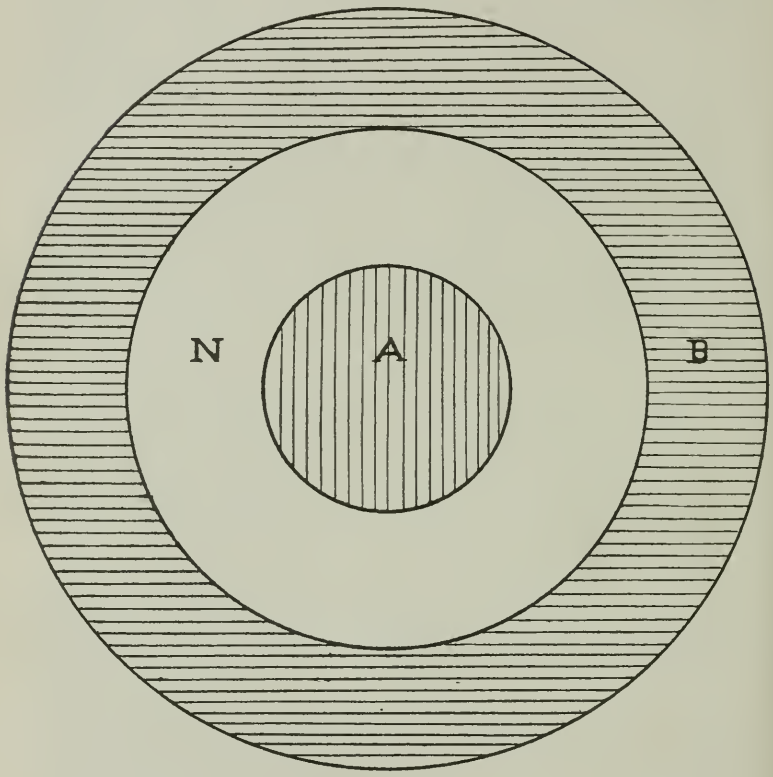
During fixation there is induced in N the colour disturbance A' and also the disturbance B'. The disturbance over the area N is therefore (disregarding meantime the result of the interaction between A and B) the resultant of $A' + B' + N$, and consequently the disturbance on the cessation of both objective and induced stimuli, that is, the disturbance corresponding to the after-image, is the resultant $A + B + N'$. This again is modified by the reciprocal inductions of A and B.

It would carry me too far were I to attempt to point out the analogy between this so-called induction and certain electrical phenomena. For the present I need only suggest that colour induction is only one of the points connected with vision which indicate a close

analogy in all links, with the exception of the final one, with electrical energy.

[*Note.*—On account of induction it is necessary, not only to rest the eyes before fixing a coloured surface whose after-image is to be studied, but also to exclude

Fig. 6.



any image of surrounding objects. This can be done by looking at the surface through a darkened tube. At the same time the disturbing influence of light passing through the sclera is obviated.

It is easy to make the visual angle subtended by the surface any size that is desired. At the end of the tube, which is held next the eye, there should be an aper-

ture of, in general, constant diameter so as to exclude the effect of difference in the size of the observer's pupil. A convenient size for this is 5 mm. The size of the opening at the other end of the tube or the length of the tube, or both, must admit of being altered to allow for different sizes of field being taken in.

The nature of the experiments in question does not call for fine gradations in the size of the fields examined, except perhaps for images which do not fall outside the macular area. I have found that four different angular apertures of about 2°, 5°, 10°, and 20° are sufficient. These can be got by alterations in the size of the opening at the far end of the tube.

I have not experimented much with smaller angular apertures than 2°. For this purpose both a smaller opening at the eye end of the tube and a means of altering the length of the tube would be desirable. The results obtained from experiments with such small fields are, so far as my limited experience of them goes, indecisive.

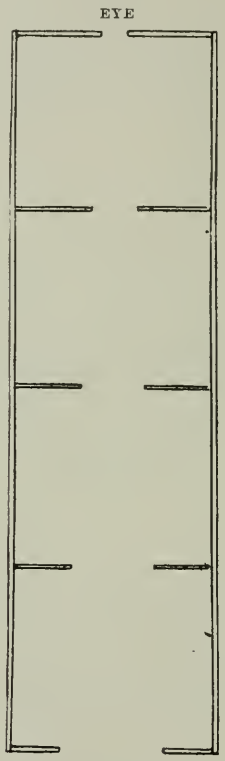
Within the limits of experimental error the angular aperture of the field visible through the tube may be taken to vary inversely as the length of the tube and directly as the sum of the diameters of the openings at its ends. Thus, supposing the field viewed had an aperture, for any particular size of opening in a tube 6 in. long, of 10°, the field would be reduced to one of 5° aperture, and consequently the area of the retinal image to quarter of its former size, by making the tube 1 ft. long. Again, with a certain definite length of tube, with the opening at the eye 5 mm. in diameter, and that at the other end also 5 mm. if a 5° field were taken in, this would be increased to one of 10° aperture by making the latter opening 15 mm. in diameter, and at the same time the area taken in would be four times as great.

The relations between tube length (L), aperture of field (a), radius of pupillary aperture (p), and that of the end opening (d) are given by—

$$a = 2 \tan^{-1} \frac{p+d}{L}, \quad L = \frac{p+d}{\tan \frac{a}{2}}, \quad d = L \tan \frac{a}{2} - p.$$

In order to study the condition of central disturbance in the area surrounding the image taken in by the darkened tube it is important to secure a complete absence of

Fig. 7.



stimulation in that surrounding area. There must, therefore, be no reflection of light from the sides of the tube. For this purpose I use a wide tube provided with a number of diaphragms in which the openings along the axis of the tube have the same ratio throughout to the length of the tube at the different distances at which the diaphragms

are placed. The sides of the tube and both surfaces of the diaphragms are coated with lamp black. Such a tube is represented in section in Fig. 7. The tube must be maintained in a fixed position, and a definite point of the coloured surface fixed in making an observation.

Under these conditions it is easy to satisfy oneself that the central area surrounding that stimulated by the coloured surface has, during the fixation of that surface, been the site of a disturbance which is complementary to the colour of the surface fixed. In after-image it appears of the same colour as that fixed, only much lighter and fainter and with no well-defined margin. It extends over an area many times greater than that taken in by the tube, and fades away gradually towards its periphery. Practically the same after-image colour impression is produced if a patch of "blackest black" is fixed on the coloured surface, with this difference only, that the colour of the after-image of the black area is equally distributed, and its margin sharply defined where it meets that of the after-image of the coloured area.]

I have made no detailed reference to retinal adaptation. The subject, though an extremely important one, is, as it appears to me, only indirectly of interest in connection with colour-vision. Indeed, it seems not improbable that it is the absence, or comparatively rudimentary character, of foveal adaptation, that accounts for the marked degree of constancy which exists as to colour impressions, so far as hues are concerned, under varying degrees of illumination. I think there is good reason for assuming that the special photo-chemical activity of the visual purple acts as the stimulus to peripheral light sensations in the dark-adapted eye. It is also probable that it does so by its effect on the rods alone. This one may infer from the fact that on dark-adaptation the increase in the photo-chromatic interval is practically due to an increased sensitiveness to neutral light impressions only, the *minimum visibile* for colour

remaining unaltered. But whether the whole change which occurs in dark-adaptation is due to the greater available quantity of the visual purple or to heightened excitability of the rods as well, does not appear to be deducible from known facts. One circumstance which is suggestive of the former being at least the more important is that that there does not seem to be any very decided extension of excitability to the fovea from the immediately contiguous parts of the peripheral retina. Prolonged rest and passivity of the end-organs and central visual cells does not appear to have any qualitative effect on colour sensation.

We may finally consider what explanation can be given for the low luminosity of the hues at either end of the spectrum. The much reduced power of discriminating between adjacent hues in these regions also calls for discussion.

It is clear that neither what has been called "the energy of luminous radiations" and also "the mechanical equivalent of light" nor the ordinary photographic effect of light can account for the former. The first is relatively high at the red end, and the second at the violet end, of the spectrum. Although, when once the threshold of luminosity has been reached, increased intensity of light causes increased luminosity, there is no other intimate relation between luminosity and the energy of the radiation. For given degrees of intensity there appear to be individual differences in response to the action of monochromatic rays from the two ends of the spectrum. As the change in luminosity is gradual until all light sensation ceases this difference has the effect of causing a difference in the length of the visible spectrum. Just as there is an individual difference in the shrillest or highest note that the ear can detect so there is a difference in the longest or shortest light waves which are capable of eliciting any light sensations. The analogy, however, only extends to the threshold of perceptibility,

which in either case is what is subject to individual differences.

If photo-chemical changes in the retinal pigment-cells are the immediate stimulus to the cones in their function as colour-sense end-organs these changes cannot be quite similar to those which have a photographic action on a sensitised plate. This is evident from a consideration of the spectrum luminosity curve. If the stimulus is photo-chemical at all and not primarily, instead of indirectly, photo-electric, the greatest chemical change must occur from the action of the rays which have the greatest luminosity effect. That it is primarily chemical so far as a neutral effect goes, seems probable from the difference in the luminosity curve for the light-adapted and for the dark-adapted eye. The low stimulus value of rays at either end of the spectrum is, therefore, probably dependent upon the smaller chemical change to which these rays give rise, before they cease to produce any effect at all, and become consequently invisible. The change that takes place in stimulus values is gradual. It is only abrupt when it falls below the light perception threshold.

As regards hue differentiation, this seems, to some extent, to depend upon luminosity and the relation which luminosity has to saturation. There are also individual differences in this respect, and that not only at the ends, but all over the spectrum.

If we exclude, however, the ends of the spectrum, for which the smaller stimulus values introduce complications, we find the greatest power of discrimination, subject only to individual differences in degree and exact position, is for the yellow and the blue, while somewhere in the intervening green discrimination is much less. That is to say, that the parts of the spectrum for which the perception all over the field of vision is most perfect are also those for which there is a greater power of differentiating between slight changes in hue. On the other hand, for that part of the spectrum for which, both for a

peripheral portion of the normal field of vision and for congenital colour-blindness, there is a diminution or absence of any qualitative impression, discrimination is normally less delicate.

This appears to me to suggest that the molecular disturbances, which I have assumed take place in the central visual cells, are physiologically less free or less isolated in some "direction" than in others. The "direction," too, in which this restriction exists is the same as that in which, for the cells connected with a particular zone normally, or with the whole retina in congenital colour-blindness, I have assumed that there is more or less complete absence of disturbance in response to physical stimuli.

Another thing which seems to indicate that there is normally less freedom for the disturbances in the direction of, or of the nature causing, a green sensation, is that on prolonged fixation green, according to its particular hue, gradually comes to appear either more yellow or more blue.

I have not tested experimentally whether any difference in acuteness of discrimination occurs subjectively, *i. e.* as after-images to their complements in the spectrum, in the case of hues near either end, as compared with the differentiation which is possible when these hues are evoked by monochromatic light.

The exact position for which there is a maximum degree of want of freedom of molecular disturbance in the central visual cells probably varies normally to some extent. This seems to be indicated by the phenomenon first discovered by Lord Rayleigh, and to which the inappropriate name of anamolous trichromatic vision has been given. The same differences in this position would also account for differences met with in that of the particular spectral colour which produces a neutral effect in the colour-blind.

To resume shortly: The chain of events leading to

colour perception, according to the views which I have endeavoured to put before you, may be taken to be something like the following:

(1) Photo-chemical action of rays on the contents of the retinal pigment-cells, possibly combined with more direct light action on the retinal cones. Thereby,
(2) Stimulation of a dual nature of the cones.
(3) Nerve current conduction from the double cone stimulation by the optic nerve to
(4) Central visual cells, in which the combination of the two factors causes different molecular disturbances, which are
(5) Psychically resolved into neutral and coloured sensations.

Further conclusions that may be drawn from the facts and considerations which have been incidentally, or more or less fully, referred to, are:

(1) That the existence of fundamental colour sensations is improbable.
(2) That the transition from polychromatism to dichromatism is more easily explained than on the assumption of a lost or crippled fundamental sensation.
(3) That physiological and congenital dichromatism are essentially the same in causation.
(4) That the molecular disturbances in the central visual cells are the same whether evoked by physical stimuli or originating subjectively.
(5) That there is a constant interaction taking place between the molecular movements in these cells in contiguous areas, an interaction known as induction, which seems to be independent of the manner in which the disturbances are evoked.
(6) That the relation between neutral and coloured light perception, though intimate, is not inseparable.

(7) That the retinal rods and the visual purple are probably unconnected with colour sensation.

(8) That the psychical processes evoked by molecular changes in the visual centre are subject to other psychical conditions, which may falsify judgment.

I should far exceed the limits of a single lecture were I to introduce any criticism of the two rival colour theories. Something of what may be advanced against both may be gathered from points that I have discussed more or less fully.

The examination of a number of intelligent, educated, colour-blind individuals impressed me long ago with the inapplicability of any theory which postulated the existence of fundamental colour-sensations. I have therefore always questioned the validity of the view that vision could be either trichromatic or tetrachromatic. I hold that it is, correctly speaking, polychromatic, and that a reasonable explanation of colour-blindness must account for the fact that polychromatism becomes in that case altered to dichromatism, with the visual functions otherwise unimpaired.

It is just in attempting an explanation of colour-blindness of the usual type that the three-colour theory appears to me to break down. Hering has made the very suggestive statement that "were it not that physiologists had obtained the three-colour theory as a venerable inheritance, they would certainly not have deduced it from the study of colour-blindness."

On the other hand, the numerous investigators who have given their adherence to the Young-Helmholz theory, the most prominent of whom is Sir William Abney, have been led, in their efforts to support it, to add greatly to our knowledge of the various phenomena of colour-vision. It must be admitted, too, that much of the information that has resulted from these investigations is difficult to

explain satisfactorily. The theory has been of great value in stimulating research by most competent scientists.

Hering's theory has also had many, though perhaps less powerful, supporters. It suffers in comparison from being less elastic, and therefore less easily forced into line with all the facts that have to be explained. On the other hand, the explanation which it offers for normal colour-vision can more consistently be applied to colour-blindness as well. It, too, has stimulated research and been productive of valuable information. Hering's own researches have been novel and suggestive, and have, moreover, had a bearing on physiological problems other than those of colour-vision alone.

Both theories cannot, of course, be right in the sense that they afford a complete and true explanation of the phenomena of the sense of colour. Yet both may contain much that may approximate to the truth, and both by stimulating investigation may still throw further light on what is yet obscure.

On the other hand, though their value in this respect may be conceded, I venture to suggest that it is important to remember that they are only helpful—not even necessary—hypotheses, in no way comparable, *e. g.* to the lunar theory, or the theory of universal gravitation, or the undulatory theory of light.

Not to recognise this is, I hold, a danger—a danger which appears, too, in by far the greater part of the work which they have inspired. The result has been, and must be, to fetter judgment as to the bearing of any new facts which are elicited on any other possible, less restricted, more comprehensive, explanation of the nature of the colour-sense than we have yet attained to.

III. DISCUSSION ON CONCUSSION INJURIES OF THE VISUAL APPARATUS.

Opening Paper.

By A. W. Ormond.

I have been asked by the Council of the Ophthalmological Society to open a discussion on "Concussion Injuries of the Visual Apparatus," limiting my remarks to "contusio bulbi," and leaving the effects upon vision of "contusio cerebri" to other speakers.

I should like, first of all, to thank the Council for the honour they have conferred on me, and to assure them that my only anxiety is to discharge the duty laid on me to their contentment. It is true that the accident of circumstance has brought a large number of these cases under my care, both at the Second London General Hospital and also at St. Dunstan's, but I am conscious that I have been too busy with routine work to find time for that careful consideration of all the problems arising out of my cases which both their number and complexity demand; at the same time I have done the best I could with the time at my disposal, and I hope they will overlook my numerous shortcomings.

On referring to the dictionary I find that "concussion" denotes a condition introduced by a more or less violent shaking of some particular organ, whereby serious symptoms may be introduced, but no *definite* lesion can be detected to account for them. We must, however, recognise that owing to the enormous power of the explosives used in modern warfare, violent shaking or concussion

may lead to very definite lesions, which may be demonstrated to be due to the concussion.

The term "contusion" designates merely a bruising, without breach of surface, so that my remarks will refer to those injuries which are the result of vibrations conveyed to the tissues from a distance, and will not include those in which a definite rupture of the globe has occurred. We shall have to discuss, also, the results of both direct and indirect bruising of the eyeball.

The globes are placed within two bony cavities, and are indirectly connected with the bones of the skull and face for the larger part of their circumference. Vibrations or oscillations which are sufficiently strong can, and do, reach the inner surface of the eyeballs—transmitted through the bones of the skull, so that heavy pieces of metal or other hard material striking the bones of the face on one side, start vibrations which are conveyed through the soft tissues to the inner tunics of the eye on the same, or even on the opposite side; similar blows on the margins or sides of the bony orbital cavities can also be transmitted through the sclerotic, and produce serious concussion or shaking of the retina or choroid. Further, that part of the wall of the eyeball which is unprotected by bone may be influenced by air vibrations, and many cases of serious damage to the globe have been recorded as due to "windage," in which, as far as the evidence goes, no solid particles have struck the face—only a force due to pressure of air or gases.

Regarding these concussion injuries I may quote Mr. Nettleship: "These indirect lesions are, it seems, to be explained by assuming that, at any rate in the case of the soft tissues, which during life are almost fluid, the force of the projectile is partly changed into vibrations of the tissue particles, which vibrations radiate in lines from the long axis of the bullet-track, so forcibly that, as Prof. Stevenson, of Netley, puts it—'they act as secondary missiles.' We must suppose that such lines of intense vibration, impinging on the outer surface of the eyeball,

though unable to cause disintegration of the dense sclerotic, can pass through it with sufficient force to produce rupture of the much less firmly knit choroid and retina immediately beneath. It is also probable that dragging on a muscular insertion by the bullet may produce an effect on the subjacent parts. That such radiating vibrations of an incompressible fluid are able to cause violent solutions of continuity in neighbouring solids has been shown experimentally by Coler (quoted by Stevenson), and his experiments have been repeated and verified by Stevenson. Vibration lesions are, I believe, well known to occur in other parts than the eye, and they are, I suppose, included in the so-called 'explosive' results of certain bullet-wounds; and it appears that the vibrations may, under some conditions, pass harmlessly through a dense structure, even through bone, and yet cause destructive changes in a soft tissue, such as the spinal cord beyond it."

The simplest effect of contusion of the eye is probably the subjective one which we recognise as "seeing sparks." Many of us can remember in our boyhood fights getting this unpleasant sensation, and later in life, in striking the front or back of one's head against a hard substance, such as a door, table, etc., one had the same experience of rapid and momentary "sparking" of the eye. This is probably the result of a sudden interference with the circulation.

From these slight and transitory effects we traverse numerous degrees of severity, until we get to results so severe that the whole globe is entirely and irrevocably disorganised.

Concussion or contusion effects may be produced in four different ways:

(1) By vibrations, the result of metal, etc., traversing the orbits, without rupturing the globes. The "through and through" wounds.

(2) By vibrations, the result of blows upon the bones of the orbit and face transmitted to the interior of the eyes.

(3) By vibrations, transmitted to the deep parts of the globe through the corneo-scleral tunic without actual rupture of the eyeball, as in direct blows on the eye itself.

(4) By vibrations, the result of air-pressure or "windage."

It will perhaps make my remarks clearer if I follow the synopsis which has been published and treat of the various structures of the globe as they have been affected by concussion injuries, illustrating as far as I am able from cases which have come under my care.

The lids and conjunctivæ seem to escape any very obvious damage, owing, doubtless, to their mobile and expansible character. Beyond some conjunctival ecchymosis, I have not seen any damage that could be attributed to concussion; on the other hand, I have been assured that the explosive effect of some of the modern shells is such that not only have the lids been torn away, but the whole eye dislocated by the mere force of the displaced air and expanding gas, and I have talked to an officer having no trace of the remains of either eye, who assured me that the lid of one eye and the whole eye itself was torn from its socket by "windage"; a shell having exploded just over him, and that no metal, earth, or solid substance touched him. It was certain that no scar of any extent was present when I saw him, only the depression covered by skin where his eyes had been, the other eye having been so damaged that its remains had to be removed. I have had several cases with a very definite and circumstantial history that no metal or solid material had touched the eyes and that the explosive force of the shell only was responsible for the damage done; in most of these there was evidence of rupture of some of the vessels of the iris and ciliary body, usually of a comparatively slight kind, but in one instance, Case No. 12, very severe and extensive hæmorrhage occurred, resulting in almost complete blindness, and the man is now under my care at St. Dunstan's.

Interesting in this connection is Case No. 57. This man was at Arras, where, two days after Christmas, 1916, the Germans bombarded from 10 p.m. to 3 a.m. He was about 150–200 yards from the bursting shells, in a solid building having shutters, but no windows. After the bombardment he fell asleep, and on awakening about 8 a.m., noticed a mist over his left eye ; this proved to be due to an intra-ocular hæmorrhage ; was this caused by "windage," or was it mere coincidence ?

It is sometimes very difficult to make up one's mind as to whether an injury is due to air-pressure or not. A soldier will often tell you that "nothing struck his eye," and that he "had no wound," but if you question him you will very often find that there were slight scratches about the face or scalp, and sometimes one can also detect small corneal opacities, which no doubt have been due to minute foreign bodies reaching the globe itself. On the other hand, the grossness of the deep changes in the eye do not suggest that these minute fragments alone produce the injury, and it seems more probable to attribute it to "windage," which is sometimes sufficiently powerful to blow a man right up in the air—a distance, often many feet—or to throw him violently on his back from the erect position.

Pagenstecher reported in 1915 two cases of injury to the retina by increase of air-pressure from grenades exploding in the near vicinity. The macula was clouded and the foveal reflex blotted out, the result, he thinks, of a delicate exudation taking place in the region of the macula. Those who have an opportunity of seeing these cases very early, which, unfortunately, I seldom have, tell us that the retina shows some pallor, and there is interference with the field of vision. What I think I have noticed is a condition in which the normal stippled appearance in and around the macula is absent and the ground work appears to be unusually uniform ; when this clears away, the retina may resume its normal appearance.

On the other hand, I have certainly seen one or two cases in which there has remained permanent mottling in the macular area with central scotoma.

A French writer has estimated that the pressure produced by a bomb explosion may be equal to as much as 10,000 kgrm. to the square metre, and he thinks, where men have been actually killed (sometimes fixed in positions in which it is probable that death was absolutely instantaneous), that it is due to a sudden freeing of air and carbonic acid gas from the blood, consequent on a sudden alteration in the pressure to which the blood is subjected, and likens the process to those changes which are found in caisson workers.

This theory seems to me to be very involved, and has been criticised by General Stevenson, who thinks on the other hand that the abnormal pressure produces concussion of the brain and interference with the functions of the nerve centres essential to life.

We need not pursue this theory further, but I should like to keep before your minds the enormous force that is generated by the explosion of modern armaments. I am, however, from my experience, and such evidence as I have been able to obtain, disinclined to accept the view that the effects of "windage" alone, apart from all injury due to solid particles, can produce extensive discontinuity of tissue. That intraocular hæmorrhage can occur from it I think probable, but the tearing of large masses of tissue and dislocation of the globes themselves, is not, I think, definitely proved.

I have seen two corneal conditions due to contusion, viz.: Striate keratitis, Case No. 69, and blood-staining of the cornea, Case No. 55. The striate keratitis was associated with lowered intraocular tension, and as it disappeared in a few days and the tension also increased, I thought it probably the result of rucking of Descemet's membrane.

Blood-staining of the cornea has been noted after concussion of the eye following on very extensive intraocular

hæmorrhage. I have only seen one case of this, but it was a particularly good instance; the discoloration itself involved the whole of the cornea and appeared to me similar to a dense and uniform 1. K.; the margin of the cornea cleared slowly and the condition being associated with a dense mass of blood in the anterior chamber, left one in no doubt as to its real nature. The colour was greenish, with a slight browny tinge in places, and was present for at least three months.

There is still a fair area of opacity left, but it is slowly getting smaller and smaller, and will, I hope, gradually disappear altogether.

Mr. Treacher Collins has investigated this subject with great care, and has come to the conclusion that the discoloration is mainly due to hœmatoidine, but is sometimes associated with hœmosiderin; that the pigment enters the substance of the cornea in the soluble form of hæmoglobin, and is there transformed into a salt not soluble in the fluids of the cornea; which it enters probably by a process of diffusion through Descemet's membrane.

Concussion changes affecting the iris and ciliary body.—A rupture of the sphincter pupillæ frequently follows both direct and indirect injury to the eye. The fibres of the muscular layer may be over-stretched, causing a permanent irregularity of the pupil, or actually torn, producing notches in the edges. The separation of the posterior pigment layer from the overlying stroma of the iris, as described by Coats, I have also seen once or twice.

A localised retraction of the iris, due probably, as was suggested by our President, to a rupture of the ciliary body, is also not infrequently seen, and in fact most of the changes in the iris that have been described as resulting from concussion and contusion have been present among my series

Von Arlt describes the mode of formation of iridodialysis as follows: "The cornea being momentarily

flattened, the circle described by the corneo-scleral border and the ciliary body with which it is firmly united is extended, whilst the sphincter iridis is contracted. The serrated connection of the iris with the ciliary body is torn to a greater or less extent, and a second communication is formed between the anterior and posterior chambers."

Fuchs thinks that there is another important factor to be remembered; he says that the blow which strikes the cornea and flattens it out also pushes the aqueous backwards; the latter tends to recede, pushes against the posterior wall of the anterior chamber, which in the area of the pupil is formed by the lens and in the rest of its extent by the iris. The latter, when pushed backwards, finds a support in the lens, except at its marginal portion, which lies outside the margin; here the posterior chamber is deepest, and is bounded only by the feeble zonular of Zinn.

The periphery of the iris, therefore, forms the most yielding spot in the posterior wall of the anterior chamber, and is the first to give way before the pressure of the receding aqueous.

Irido-dialysis, however, may be the result of an injury to the posterior half of the eye as in Case 19, where the bullet went through the apex of the orbit.

Defects of accommodation are not uncommon as result of shell shock, and the manifestation of errors of refraction, previously unsuspected by the soldier, has led to the improvement of the vision of many cases by the correction of astigmatism and hypermetropia with suitable lenses.

An interesting condition of hippus (Case 71) is recorded; this officer had no p.l. in one eye and very slight vision in the other. Both eyes showed the condition of hippus.

Concussion changes affecting the choroid and retina.— It has long been known that both concussion and contusion injuries, direct and indirect, are able to damage the retina and choroid. There is little to be added to

General Stevenson's account, published in 1897, and to Mr. Nettleship's observation already recorded in the *Transactions* (vol. xxi), except perhaps to enlarge our ideas as to the distance over which these vibration effects can be carried.

At a discussion which was held in this Society in 1915, Colonel Lister stated that as far as he had noticed, if the bullet did not go through the orbit itself there were no concussion changes in the fundus; the orbital walls might be cracked or broken by some accident, yet the fundus showed no ophthalmoscopic change; whereas if the bullet passed through the orbit, the globe not being ruptured, the usual dramatic concussion picture was seen. This, however, must be very seriously questioned.

In these accidents, various sized pieces of metal are suddenly and violently driven in all directions. Some are, no doubt, minute fragments; some may be sufficiently large to actually tear the head from the body.

I have no hesitation in saying that it is not necessary at all for the metal to traverse the orbit to produce the dramatic concussion changes referred to by Colonel Lister.

A French surgeon, Dantrelle, has recently drawn attention to what he describes as a "dangerous area," and he does not mean the space immediately surrounding the corneo-scleral margin.

Dantrelle's "dangerous" area is really the margin of the orbit for a distance of about two centimetres backwards from its edge, and he gives it as his opinion that a blow of sufficient force striking the margin of the orbit invariably leads to serious permanent damage to the globe itself, and my experience entirely agrees with this.

The large majority of the cases that I have seen during the last year showing ruptures of the choroid and retina, with extensive intraocular hæmorrhage, have been due to damage done to the margin of the orbit; and if a man presents himself with the evidence of a wound, either at the external angular process or in the malar bone, or at

the root of the nose, or even on the opposite side of the face, one finds almost invariably extensive damage to the globe, even though the eye may appear on superficial examination to be untouched.

Another condition not infrequently seen as the result of a shaking of the eye is a widely spread disturbance of the retinal pigment. It is difficult to say what is the actual pathological factor which causes the precise allocation of this pigment. Sometimes it appears as grains of black pepper scattered over a mottled field, sometimes as triangular or spindle-shaped bodies with processes simulating retinitis pigmentosa. Sometimes, again, it is collected into one or two definite places, where it forms dense masses of pigment without any sign of the choroid being involved.

I think that sometimes a rupture of the retina takes place on a flat plane, a large area of retina being temporarily displaced by vibrations, which cause a sliding movement of the inner part of the retina over the pigment layer which is attached firmly to the choroid. This sort of movement, of one surface on another, takes place as the result of an oblique glancing blow, the retina being shifted at its most vulnerable part, viz., the bacillary layer. The alternate hypothesis is that the pathological lesion is the result of section of the posterior ciliary vessels, and certainly in some of my cases this is extremely likely to have occurred, as the bullet has passed through the orbits behind the globe, but close to the nerve-head. The interference with the choroidal circulation leads to a proliferation of the pigment epithelium, some of which is carried into the retina by leucocytes.

Hirsch pointed out in 1905 that this condition not infrequently follows severe contusion of the globe, when the picture of retinitis pigmentosa may be closely simulated.

It is, however, more common to find in association with this fine retinal pigmentary change a definite rupture of the choroid.

Further gross changes in the choroidal and retinal vessels produce definite hæmorrhages, varying in size and quantity up to an entire obliteration of the whole of the red reflex.

The pathological changes found in cases where the retina and choroid are ruptured have been described by Mr. Nettleship in the twenty-fourth volume of the *Transactions* of this Society :

"The choroid is more easily ruptured than the retina—that is to say, the choroid may be found ruptured alone, without the retina being involved. We often see this clinically, in cases where the retinal vessels can distinctly be seen crossing over the ruptured area. On the other hand, it is not very clear whether the retina alone is often ruptured, but one can say definitely that many cases present themselves with retinal hæmorrhage, in which subsequently no evidence of involvement of the choroid can be found clinically. It is probable, therefore, that the choroid and the retina may be ruptured singly and alone. On the other hand, it is frequent to find both these membranes involved. When this occurs, there is more or less complete fusion of the torn edges to each other, and to the sclera and vitreous, by adhesive inflammation. This explains the comparative infrequency with which one finds detachment of the retina as a result of the traumatism resulting from concussion."

The hæmorrhage, when it occurs, takes place in the layers of rods and cones—that is, the bacillary layer of the retina between the pigment epithelium and membrana limitans externa of the retina. This area is the one most easily disintegrated, and as a rule the layer of retinal pigment-cells adheres to the choroid, of which it really is a part. On the other hand, the membrana limitans externa is merely the boundary line of the sustentacular tissue of the retina. We therefore, not infrequently, find the retinal vessels lying distinctly in front of extensive hæmorrhages. When, however, the retina itself is also torn, this blood passes through the gap, and spreads out

between the membrana limitans interna and the hyaloid membrane of the vitreous. No hæmorrhage is usually found between the choroid and the sclerotic.

The presence of blood appears to have an irritating effect upon the surrounding tissue when it is extravasated in this way, and produces a low form of plastic inflammation in the situations where the hæmorrhage has been shown to exist. This produces a welding together of the various structures involved, with consequent atrophy of the tissues that are entangled, and in this plastic inflammatory material, after a period of some months, new fibrous tissue appears, which seems to be entirely derived from the choroid—that is, becomes organised and forms dense white masses, which subsequently undergo a certain amount of shrinking. The simplest form of rupture of the choroid seems to be produced by a tear through the lamina vitrea and pigment epithelium; a small fold of retina is lifted up by the exudation of blood.

I have often noticed pear-shaped masses of hæmorrhage, globular in form, pushed forward into the vitreous, and showing no tendency to separate laterally or become diffused with the surrounding tissue. It appears to me that these hæmorrhagic masses are surrounded by a membrane which keeps them in bounds, and probably the hyaloid membrane in these cases is not ruptured, but only pushed forward in front of the hæmorrhagic mass. All the recorded cases suggest that a low form of inflammation results from the presence of large intraocular blood-clots. This, I take it, is due to the irritating effect produced by the presence of blood itself, as clinically one has no evidence whatever of any inflammatory trouble being present, the eye showing no increased redness and the patient not complaining of any pain.

One often sees small wounds of the conjunctiva covered by the lower lid, and on examining the interior of the eye, finds definite hæmorrhage in the extreme anterior or lower periphery of the globe. This, no doubt, is due to direct hits upon the eye, but they have not ruptured the

sclerotic, though they have produced a sharp blow on the superficial surface of the eye, leading to a tear of some of the vessels in the ciliary region or just behind it.

I have had numerous cases of this sort, and they are very easily overlooked, as the patient's vision is seldom affected. Sometimes one is led to search for them by the history that there was some blood found in the eye when first seen, and these injuries are not infrequently associated with hyphœma (*vide* Case 50).

By a "through-and-through" wound is meant one in which a bullet enters on one side of the head and passes through the orbits, leaving on the opposite side. Sometimes the bullet enters through one orbit, destroying the globe in its path, and passing obliquely downwards, stops in the maxillary antrum on one or other side. On several occasions, earlier in the war, I removed metal from the antrum, but these "through-and-through" wounds are not so common now as when the sniper had a freer hand, as in 1915. They usually produce extensive intra-ocular hæmorrhage, with rupture of the choroid and retina; sometimes the optic nerve is directly damaged, and sometimes the iris, ciliary body or lens are indirectly involved.

In the majority of these cases the line the bullets take is the line of least resistance—that is to say, they traverse the skull where the bones are thinnest and the air cavities most numerous. The exit or entrance scar is usually in an area bounded in front by the external angular process, above by the temporal ridge, below by the zygoma, and behind by the temporo-maxilla articulation; in other words, the temporal fossa and the line of their passage traverses the orbits, posterior ethmoidal cells, and the middle or superior meatus of the nose. Owing to the proximity of this line to the base of the skull and the not infrequent presence of "starred" fractures, there is no doubt that these "through-and-through" wounds are far more dangerous to life than almost any other of the wounds leading to loss of sight, and if these men's subsequent history is known it will be

found that many of them succumb to cerebral abscess, meningitis, or end in a lunatic asylum.

I can recall at least half a dozen cases where death from cerebral abscess or meningitis has supervened sometimes twelve or eighteen months after injury, and these wounds are, in my opinion, much more dangerous than the condition of the patient or appearance of the wound leads one at first to suspect.

Detachment of retina.—Instances where the retina has become detached are not so infrequent as the opponents of the traumatic origin of retinal detachment would expect. Cases 7, 32, 58, and 79 are cases where, after a varying lapse of time, the sight failed owing to detachment supervening. Two of these four cases were myopic, and all were the result of indirect injuries. The remaining Cases 23, 53, 66, and 76 were examples of solid detachment due to blood being poured out in the layers of rods and cones and pushing the retina forwards.

Concussion cataracts.—The examples of concussion cataracts recorded in my notes have been extremely interesting and instructive. Most of them have shown the stellate form of posterior cortical cataract, and have been associated with dislocation of the lens or irido-dialysis, the result of concussion. A smaller number showed anterior changes.

I might quote Mr. Hudson's description of the posterior cortical variety: "Within a few hours of the receipt of injury there appear in the region of the posterior surface of the lens feather-like opacities whose precise orientation is dependent upon the architecture of the lens fibres in relation to the lens star, and exhibits accordingly some variations in individual cases. These feather-like opacities are first seen in the central region, whence they tend to extend outwards towards the periphery of the lens, to become continuous with a fringe of short radial striate opacities disposed in conformity with the long axis of the equatorial fibres, and also appearing at an early date.

"At a slightly later date vacuoles may, in some cases,

be observed in the midribs of the feather-like opacities, between their rays, and between the peripheral striæ. Usually within the next few days the character of the opacity changes, the striate formation being replaced by a rather dense lacework pattern in which, as a rule, few definite vacuoles can be recognised with certainty, but in which the radial plan may still be traced. This in turn is succeeded by a definitely vacuolar pattern, and at this stage not infrequently rather large vacuoles, either discrete or in a palisade formation, and having their axes in conformity with that of the lens fibres, may be observed in the equatorial region of the lens. The vacuolar pattern next becomes more open, as though from the formation of larger vacuoles by the coalescence of smaller ones.

"In the later stages the central figure becomes broken up into discrete vacuoles, which become progressively smaller in size, until finally all that remains visible is a collection of fine granules, which may by their arrangement afford some suggestion of the antecedent stellate figure."

The changes that I have seen have been mainly vacuolated and in some instances the peripheral striæ also have been present. I think I have satisfied myself that these manifestations of concussion are undoubtedly temporary. In one case the centre of the star seemed to open out so that a ring was formed and the radiations could be seen passing from the outer part of the ring, the centre becoming quite clear and the vision improving accordingly.

The posterior cortical changes supervene soon after the lens has been damaged; Mr. Hudson found them present in some cases a few hours after the injury. The vacuolation appears after the striation in from three days to a week. The lacework patterns are merely complex vacuolations differing only in degree and arrangement of the vacuoles; the granules are the remains of the vacuoles. We may, therefore, simplify the description of the changes by saying that they are vacuoles in various stages of

development and arrangement, except in the very early condition, when they are striate and grouped in relation to the lens star at the posterior central part of the lens, and also at the equator.

The views put forward to explain the condition are that they are due to distended physiological lymph spaces in the lens, or to definite but usually transitory changes in the lens fibres themselves, but I do not agree with either of these suggested causes. The first is improbable because Leber has disproved the existence of lymph spaces in the lens; and against the second it may be urged that if the lens fibres themselves were affected we should have more frequently the generalised opacity of the lens starting from the posterior surface, whereas these stellate opacities either disappeared entirely, leaving the lens quite clear, or the lens turns opaque in some other plane, not originating at the posterior surface. I think the change is not in the lens, but rather on the posterior lens surface, and is due to fluid coming from the vitreous passing under the posterior lens capsule, caused by a slight movement of the lens itself in the hyaloid cup of the vitreous body.

Instances of the anterior opacities have also been seen; an officer, wounded at the beginning of this year, showed very well the condition that was termed by Vossius "concussion ring." This man was wounded December 10th, 1916, and I saw him on the 22nd—that is, twelve days after injury. The right eye had been removed owing to the extensive damage from a shell bursting while he was in a dug-out. The left eye, however, had only been slightly affected. Numerous pieces of stone and metal had been scattered over his face, and there were some minute scars on the conjunctiva and in the cornea. He complained of defective vision in his left eye, and was very much worried by his loss of sight. On examining the fundus I was unable to obtain a perfectly clear view, but I could see a D-shaped sub-hyaloid hæmorrhage in the lower and outer part of his field;

this is rather unusual, as it was some distance from the macular region; beyond this the fundus seemed fairly healthy. On examining him at a short distance away by transmitted light, I saw occupying the centre of the red reflex an extremely well-formed definite ring. By paralactic movement, I was able to ascertain that it was just in front of the nodal point, in the lens, but I could not see it by focal illumination at all. The ring, on careful magnification, appeared to be made up of extremely fine opaque dots, and in one or two places there seemed to be slight gaps in the opaque area of the ring. The lens itself did not show any definite opacities, and, as I say, the opacities that one could see with transmitted light were not visible by focal illumination. There was no actual pigment present, and the condition altered very slowly; subsequently the lower area disappeared entirely about 6 o'clock, leaving a horse-shoe shaped body, and the whole opacity had completely disappeared in five months.

Vossius assumed that these ring opacities were due to the pupillary margin of the iris being pressed against the anterior surface of the lens by the cornea. Hoeg's view, however, is that the ring is the result of sudden increase of pressure in the anterior chamber, which forcibly presses the iris against the lens and causes the ring opacities. The increased aqueous pressure is also assumed to be the cause of the opacities outside and within the ring. A condition that was described by Gifford, of Omaha, in the *Ophthalmic Record* for August, 1910, was also present in this case, viz. a further area, where there were innumerable fine opacities scattered throughout the lens. This further area was not circular, and was very much finer than the opacities forming the ring; in fact, I did not see them at all until the occasion of my second examination, and they disappeared quite soon after the patient came under observation. It probably corresponded to one of Captain Whiting's cases of double discoid opacity. Mr. Lawford, in the *Ophthalmic Review* for 1887, has given a *résumé* of the opinions of different

authors with regard to the formation of another type of concussion cataract. In his cases there appeared to be a tearing of the lens capsule, and I could clinically verify this condition in some of my patients. The edge of the capsule could be seen curled up on the anterior surface, and a gradual change took place in the superficial portion of the lens, producing in some cases an incomplete cataract and a complete one in others. It is improbable, however, that the posterior cortical opacities are associated with a rupture of the capsule, and the Vossius concussion ring certainly is not.

It is not true that the anterior discoid cataract is never visible by focal illumination, as in one case (No. 74) I was able to make out its position by the means of that and a binocular loupe, and it was distinctly deep to the anterior capsule, but in front of the nucleus.

As far as I have seen, these anterior and posterior concussion changes do not lead to a general opacity of the lens unless the capsule has been definitely ruptured, and even large, extensive, posterior cortical changes tend to diminish and gradually disappear.

In two men, now under my care at St. Dunstan's, who were injured nearly two years ago, I have found changes which I think are the remains of posterior stellate cataracts. They consist of oval or rounded vacuoles, each one single and isolated, arranged sometimes in groups of five to six in number, sometimes more, looking exactly like bubbles of air in badly made glass.

Treatment.—There are one or two points regarding treatment that I should like to emphasise:

Firstly, the extreme usefulness of the "hot coil," *i. e.* a pad of lint or bandage through which a graduated electric current is passed. By this means, continuous dry heat is applied for an unlimited time, and in assuaging the pain of a bruised eye it has no equal. I have had several fixed in all my wards, and they are continuously in use.

Secondly, I still adhere to the opinion I expressed last

year, that mechanical means are the best for hastening the absorption of vitreous and retinal hæmorrhage.

Thirdly, I have not hesitated to deal with the complete traumatic cataracts as soon as the eye has recovered from the immediate effects of the traumatism, and in only one case have I regretted doing so. In this case a small piece of wood was present in the ciliary body, and a septic irido-cyclitis developed, which resulted in a shrunken, blind eye.

Fourthly, in the treatment of the wounds involving the air-cavities flanking the orbits free drainage is a necessity, and ophthalmic surgeons should be cognisant of the modern methods of dealing with suppurating sinusitis, and should not hesitate to deal with these conditions under certain circumstances.

Fifthly, that hot fomentations, frequently applied, form the best dressings for wounds about the orbits, which are almost invariably septic.

In conclusion, I might suggest that the subjects about which discussion would be most useful are :

First.—The nature and extent of the lesions produced by "windage."

Secondly.—The pathology of the pigmented retinal conditions not infrequently seen as a result of concussion.

Thirdly.—The pathology of the non-progressive, often retrogressive, concussion cataracts.

Concussion Wounds of the Eyes.

Illustrative Cases.

Wounds of "dangerous area"	1, 2, 6, 7, 9, 11, 14, 16, 22, 25, 26, 27, 31, 33, 43, 45, 46, 54, 62.
Concussion cataracts :	
(*a*) Complete . .	2, 18, 21, 28, 34.
(*b*) Posterior cortical	22, 48, 63, 64, 68, 69, 70, 75, 76.
(*c*) Anterior . .	56, 72, 74, 78.
"Through and through" wounds	3, 8, 17, 20, 24, 28, 29, 30, 34, 35, 36, 37, 40, 65, 67, 70, 71.
Detachment of retina .	7, 23, 32, 53, 58, 66, 76, 79.
"Windage" . .	5, 10, 12, 15, 44, 47, 49, 57, 59, 62, 79.
Pigmentation of retina .	3, 11, 16, 25, 39, 40, 42, 49, 52, 54, 59.
Macula changes . .	3, 9, 11, 15, 16, 31, 49, 51, 80.
Irido-dialysis . .	4, 19, 41, 63, 72, 75, 77.
Damage to optic nerve .	17, 23, 24, 28, 29, 36, 61, 65, 70, 76, 60.
Dislocation of lens .	21, 22, 48, 64, 69, 77.
Injuries to extra-ocular muscles	23, 27, 38, 67.

No.	Initials.	Age	Rank.	Corps.	When, where, and how wounded.	Vision of each eye.	Condition of eye and notes in the text.
1	A—	22	Captain	6th Lincolns	17 : 9 : '16; Thiepval; bomb	R.E., nothing direct; L.E. $\frac{6}{6}$	Direct injury to right malar bone. Fracture involved nose also; not antrum. Rupture of retina and choroid concentric with O.D. Much vitreous hæmorrhage, which became absorbed by November 17th.
2	J. H. L—	?	—	12th Durham Light Infantry	7 : 10 : '16; Le Sars; shrapnel	R.E. $\frac{6}{6}$; L.E., D.L.	L.E.: Lens contused; rupture of sphincter; serrated edge to sphincter pupillæ. Commencataract. Direct wound of supra-orbital ridge and root of nose. Capsule of lens ruptured and displaced.
3	G. F. H—	24	Lieutenant	116th Machine Gun Corps	20 : 10 : '16; Thiepval; shell burst	R.E. excised; L.E., L.M.O.	L.E.: Pigmented condition of fundus at macula. Rupture of infra-temporal vessels. Intra-ocular hæmorrhage. Probably had a "through and through" wound as scar on left side in front of ear.
4	N—	—	Private	Royal West Surrey	10 : 10 : '16; Somme; gunshot wound	R.E. blind; L.E. $\frac{6}{6}$	Irido-dialysis. No scar seen. Metal removed from between the lids. No P.L. Haze of lens, vitreous, optic disc not seen. Nerve probably severed.
5	A. M. F—	32	2nd Lieutenant	7th South Lancs.	31 : 10 : '16; nr. Thiepval; air; no actual contact; "windage"	R.E. $\frac{6}{6}$ $\bar{c}$ + 1; L.E. $\frac{6}{6}$ $\bar{c}$ + 5 = $\frac{6}{6}$	"Windage." Fine haze in vitreous. Some retinal hæmorrhage in lower and outer part. Hyphœma after the accident. No evidence of direct injury.
6	G. H—	22	Private	2nd Bedfords	12 : 10 : '16; Somme; small shell splinters	R.E., L.M.O. L.E. $\frac{6}{6}$	R.E.: Rupture of choroid around optic disc. Wound on nasal side of right orbit. F.Bs. in orbit.
7	J. D—	39	Private	Royal Irish Fusiliers	15 : 8 : '15; Suvla Bay; shrapnel	R.E., perception of light only; L.E. enucleated	R.E. was not hit directly. Vision failed gradually. Large detachment of retina spreading up from O.D. Direct injury to left side of head.
8	F. H—	21	Private	Royal Fusiliers	20 : 12 : '15; by snipers; bullet	R.E. enucleated; L.E., can see contrasts	"Through and through" wound. Rupture of choroid and hæmorrhage in vitreous.

9	A. W—	29	Private	—	5 : 4 : '16; Vermelles; 2 pieces metal at different times	R.E. $\frac{6}{36}$; L.E. enucleated	Was hit twice; on two separate occasions. R.E.: Wound over zygoma. Concussion changes at macula only. Two small patches of choroidal atrophy near yellow spot.
10	A—	35	Private	7th Canadians	31 : 10 : '16; Le Sars; explosion shell	R.E. $\frac{5}{6}$; L.E. $\frac{6}{18}$	"Windage." Hyphœma. Vitreous hæmorrhage in form of blobs of blood in extreme periphery below.
11	H. W. O—	22	Lieutenant	1st Dorsets	18 : 11 : '16; Serre; bomb	R.E. $\frac{6}{24}$; L.E. $\frac{6}{6}$	R.E.: Pigmentary disturbance of retina at macula central scotoma. Tears in iris and pigment on anterior surface of lens. No gross fundus lesion beyond fine scattered pigment. A mass of metal struck his helmet, which was forced down on to right temporal ridge.
12	C—	19	Private	1st Cheshires	3 : 9 : '16 ?; Guillemont; shell burst; "windage" ?	R.E., P.L.; L.E., L.M.O.	"Was not touched." Shell burst within three yards. He did not lose consciousness, but was knocked down. "Windage." R.E. full of blood, which is turning white above. V. — P.L. L.E.: Blood in lower two-thirds of vitreous. Retina can be seen above. V. fingers 1 ft. He had, however, two small scars, one in right upper lid and one in left lower lid.
13	G. B—	22	Private	2/9 Yorks. A.	16 : 7 : '15; Kummel; shrapnel	R.E. $\frac{6}{9}$; L.E. $\frac{6}{18}$	Concussion blindness cured by hypnotic suggestion. Shrapnel burst at edge of trench and threw a lot of sand in his eyes. L.E.: Has a long wisp of scar tissue coming from vessel on outer side of optic disc.
14	W. P—	38	Private	Grenadier Guards	15 : 9 : '16 ?; "High Wood"; shell	R.E. $\frac{6}{9}$; L.E. $\frac{6}{60}$	Deep scar over left malar bone, with adherent skin and scar of trephining for depressed fracture of temporal region. Extensive rupture of choroid. Hæmorrhage in vitreous. V. indirect, in, out, and down, of field.
15	M—	41	Captain	?	15 : 11 : '14; France; shell-burst; "windage"	R.E. $\frac{6}{6}$ normal; L.E. $\frac{6}{12}$ partly	Seen by me more than twelve months after injury. L.E.: Slight but definite patch of choroidal atrophy at macula paracentral scotoma just above fixation point.

No.	Initials.	Age.	Rank.	Corps.	When, where, and how wounded.	Vision of each eye.	Condition of eye and notes in the text.
16	A—	31	Private	9th King's Liverpools	24 : 9 : '16 ; Grandcourt ; shrapnel	R.E. $\frac{6}{\frac{1}{8}}$; L.E. $\frac{6}{9}$	R.E. : Adherent scar in malar region. Fine pigmentation in region of macula extending widely. Two small ruptures of choroid in extreme lower periphery.
17	S. S. T—	32	—	2nd Canadian Mounted Rifles	25 : 9 : '16 ; Courcelette ; Somme ; bullet	R.E. enucleated ; L.E. blind	"Through and through" wound. L.E. : Optic nerve cut through and rupture of retina and choroid.
18	E. T—	21	Private	Royal Sussex	4 : 3 : '16 ; France ; hand grenade	R.E., shadows at a few feet ; L.E. enucleated	The vision gradually failed in this case, and in November, 1916, the note was T — 2. No red reflex. Shrinking mass of organised blood-clot in vitreous. Cholesterin. Bare P.L. Lens opaque.
19	A. C—	28	—	Rifle Brigade	15 : 9 : '16 ; Somme ; bullet ; "through and through"	R.E. enucleated ; L.E. blind	Irido-dialysis T — 2. "Through and through" wound.
20	J. S—	44	Private	1st Royal North Lancs.	Struck by bullet	R.E., L.M.O. ; L.E. enucleated	Probably "through and through" wound. Both temporal regions showed scar. R.E. — 1. Pupil small and fixed. Some iritic adhesions. Lens slightly opaque. Extensive hæmorrhage into vitreous.
21	S. W—	25	—	Cheshires	1 : 9 : '16 ; Thiepval ; rifle grenade	R.E. $\frac{6}{\frac{2}{60}}$ + 9 ; L.E. P.L.	Rupture of retina at macula in R.E. Iris torn at pupillary margin. Lens dislocated up and in, opaque and shrivelled. L.E. : Traumatic cataract *in situ*. Irido-dialysis. Several wounds of the skin of face.

22	T. M—	20	—	King's Liverpool	8 : 6 : '16; Malancourt; Somme; 18 lb. shell; "whiz-bang"	R.E., no P.L.; L.E., hand movements at 1 yd.	Face struck with large piece of metal; left malar bone, right supra-orbital region; eyes themselves not struck. R.E.: Iris tremulous. Lens dislocated up and outwards. Rupture of retina and choroid. Concussion cataract, palisade opacities. L.E.: Lens drawn up and out behind iris. Fundus seen $\bar{c}$ + 10 sph.; general pigmentary change all over retina. Small choroidal rupture near disc.
23	M—	18	No. 4811	Irish Guards	20 : 12 : '14; bullet; sniper; "through and through"	R.E., P.L.; L.E., no P.L.	Both eyes *in situ*. Left external rectus muscle is paralysed. R.E.: Organised mass of blood pushing retina forward. Detachment of retina. L.E.: Blind. Optic nerve ruptured at nerve-head. Probably direct injury to globe.
24	D—	33	Sergt.	—	10 : 5 : '15; Aldershot; bullet	R.E., L.M.O.; L.E. blind	"Through and through" bullet wound. R.E.: Rupture of choroid. L.E.: Complete atrophy of nerve-head.
25	K—	22	Private 50	Australians	18 : 8 : '16; Somme; splinter of shell casing	R.E. $\frac{9}{6}$; L.E., fingers at a few feet	Struck on margin of orbit on left side below cut lid. L.E.: Extensive pigmentary changes. Pigment peppered all over fundus. One or two short white streaks, showing rupture of choroid.
26	H—	29	Private	6th North Hants.	16 : 11 : '16 ?; Albert; shrapnel	R.E. $\frac{6}{6}$; L.E. counts fingers at 1 ft.	Struck on left supra-orbital margin. Rupture of choroid. Pigmented retina. Vitreous hæmorrhage.
27	T. Y—	25	Private	—	3 : 9 : '16; Beaumont Hamel; shrapnel	R.E. $\frac{6}{18}$; L.E. *nil*	L.E.: Scar on left orbital margin. Vitreous full of blood. Dull red reflex. R.E. was amblyopic. Squint developed after injury. Concomitant fixes with R.E. Vision did not improve.
28	F—	36	Lance-Corporal	2nd Leicesters	27 : 1 : '15; La Bassee; bullet	R.E. enucleated; L.E. blind	"Through and through" bullet wound. Bullet entered left side $\frac{1}{2}$ in. behind outer canthus. Left eye at outer angle of right orbit destroying the globe. L.E.: Nerve cut through. Lens subsequently became cataractous. Anosmia. Vitreous hæmorrhage.

No.	Initials.	Age.	Rank.	Corps.	When, where, and how wounded.	Vision of each eye.	Condition of eye and notes in the text.
29	R. C—	20	—	1st Royal Fusiliers	16 : 12 : '14; Armentières; bullet	R.E. enucleated; L.E. blind	"Through and through" bullet wound. Bullet entered on right side through right upper lid and frontal sinus, passing obliquely backwards to a point 1½ in. above and behind external angular process. L.E.: Rupture of optic disc and choroid in neighbourhood of disc.
30	B—	29	Sergt.	1st Rifle Brigade	27 : 8 : '14; Ligne; bullet	R.E. enucleated; L.E. L.M.O. to nasal side	"Through and through" bullet wound, which entered on left side behind outer canthus and left through right orbit, destroying right globe. L.E.: Optic disc pale. Large mass of soft white organised blood in inner side. Outer half of retina normal.
31	E—	19	Private	1st Lancs. Fusiliers	End of October, 1916; Bapaume; shell exploded and burying him	R.E. L.M.O.; L.E. $\frac{6}{6}$	R.E.: Scar over outer canthus. Scattered pigment changes at macula; grosser changes at outer half of retina. Pigmented scar tissue. Rupture of choroid there.
32	P. W—	?	Private	2nd Gordons	September, 1915; Hooge; shell exploding and buried him in mud	R.E., L.M.O.; L.E. $\frac{6}{6}$	He was buried in mud, etc., by a shell explosion September, 1915. Sight of right eye affected, but improved. He returned to trenches and noticed nothing until the autumn, 1916, when sight gradually failed. Fine pigment changes near O.D. Rupture of retina below the disc. Transparent detachment of retina, outer side. Vitreous haze.
33	C—	20	Private	1st Essex Regiment	Somme; shell struck him	R.E. P.L.; L.E. $\frac{6}{6}$	R.E.: Struck on outer wall of right orbit, where are two small scars. Extensive vitreous hæmorrhage and concussion changes on outer side.

34	W—	19	Private	1st Batt. Scots Guards	21 : 1 : '15; La Bassee; bullet	R.E. enucleated; L.E. $\bar{6}$ + 12 = $\frac{6}{60}$	"Through and through." Bullet entered at inner canthus of L.E., scarring left cornea, and passed through nose and out through right orbit. Lens of L.E. turned opaque and was needled. Organised blood in vitreous.
35	W. G. S—	?	Private	Northamptons	15 : 11 : '14; Neuve Chapelle; bullet	R.E., L.M.O.; L.E., P.L.	Some vitreous hæmorrhage in both eyes, which has organised. No details of fundus seen in either eye. Had wounds on lids and left temple.
36	C. V—	25	?	Belgian	25 : 10 : '14; Dixmude; bullet	R.E. blind; L.E. enucleated	"Through and through" bullet wound. Entered left side, striking upper margin of the orbit and traversed head to a point just in front of right ear. R.E.: Optic nerve severed close to globe; rupture of retina and choroid from optic disc, upwards and outwards.
37	S. I—	?	—	3rd Batt. Rifle Brigade	16 : 1 : '15; ?; bullet	L.E. $\frac{6}{60}$	"Through and through" bullet wound. Entered through right orbit, passing downwards and leaving through malar region. Left pupil ruptured. Vitreous hæmorrhage.
38	B—	—	Lieut-enant	—	Hill 60; shrapnel	R.E. $\frac{6}{6}$; L.E. $\frac{6}{6}$	Diplopia. Movement of L.E. upwards defective. Both eyes apparently normal. Condition lasted until July, 1915, and then disappeared. ? Hæmorrhage in muscle.
39	J. B—	41	—	A.S.C.	1 : 8 : '16; Loos; "whiz-bang"	L.E. $\frac{6}{60}$ $\bar{6}$ + 6 amblyopia; R.E. P.L.	R.E.: Rupture of choroid at macula. Very small fine pigment scattered over fundus, around the optic disc, looking like black pepper on mottled background. Scar of right lower lid.
40	B—	31	Lance-Corporal	2nd Highland Light Infantry	1 : 7 : '16; Vimy Ridge; bullet	R.E. no P.L.; L.E., L.M.O. at 8 ft.	"Through and through." Bullet entered on left side in temporal region and traversed head horizontally. R.E.: Optic atrophy. Extensive fine pigment changes. L.E.: Rupture of choroid. Pupil reacts.
41	T. L—	32	Private	1st Royal Warwicks	26 : 8 : '16; Hill 60; shrapnel	L.E. $\frac{6}{6}$; R.E. ?	R.E.: Irido-dialysis. Rupture of sphincter. Mixed astigmatism. Fundus healthy. Small corneal scar. Contusio bulbi.

No.	Initials.	Age.	Rank.	Corps.	When, where, and how wounded.	Vision of each eye.	Condition of eye and notes in the text.
42	B—	27	Lance-Corporal	Welsh Fusiliers	16 : 10 : '16; Loos; shell splinter	R.E. $\frac{6}{36}$; L.E. $\frac{6}{6}$	Fine concussion changes. Pigmented disturbance of retina and choroid at inner side. Metal removed from inner side of right orbit.
43	H. J. S—	24	Lieutenant	12th Suffolks	10 : 8 : '16; Marne; bomb	R.E. *nil*; L.E. $\frac{6}{6}$	Rupture of iris and sphincter pupillæ from blow on right malar bone. No red reflex. Blood in cornea. Separation of iris stroma from posterior pigment layer. This can be seen for three quarters of the circumference of the iris. Recurrent hæmorrhage into vitreous. Excised February, 1917.
44	C. S—	22	Corporal	18th Welsh Regiment	17 : 10 : '16; Loos; explosion of shell	R.E. $\frac{6}{9}$; L.E. $\frac{6}{36}$	"Windage." Some retinal and vitreous hæmorrhage in extreme periphery below. No scar of wound found.
45	I. J. T. N—	20	Private	Royal Fusiliers	28 : 5 : '16; Vimy Ridge; shrapnel	R.E. enucleated; L.E. $\frac{5}{60}$	Fracture of left malar bone and lower margin of orbit extending to nose. Middle turbinate bone bound down. Obstruction to tear passage. Rupture of choroid extending to the O.D.
46	G. H—	22	Private	2nd Bedfords	12 : 10 : '16; Somme; shell or gunshot	L.E. $\frac{6}{18}$; mixed ast.; R.E., shadows at a few feet	Wound of orbital margin. Rupture of choroid.
47	F. T—	17	Private	9th Norfolks	Date ?; Somme; shell explosion	R.E. $\frac{6}{6}$; L.E. $\frac{6}{36}$	"Windage." Small rupture of choroid and hæmorrhage in extreme Anterior periphery. No scar.
48	A. C—	56	Civilian	—	3 : 2 : '17; London; was cutting wood when a piece flew up and struck L.E.	R.E. $\frac{6}{6}$; L.E. $\frac{6}{36}$	Lens dislocated T. + 2. Trephined sclera. Posterior polar concussion cataract, star-shaped, which subsequently altered position, passing to periphery and diminishing in size. Contusio bulbi.

49	A. E—	18	Private	20th Middlesex	27 : 8 — ; Amiens ; explosion of ammunition	R.E. $\frac{6}{60}$; L.E. $\frac{6}{6}$	"Windage." Was blown up some feet into the air. R.E.: Concussion changes at macula. Disturbance of pigment. Central scotoma. Contraction of field to outer side.
50	G. S—	43	Civilian ; labourer	—	18 : 11 : '16 ; wounded whilst acting as game beater ; struck by pellets of scatter gun	R.E. $\frac{6}{6}$; L.E. $\frac{6}{6}$	Some vitreous hæmorrhage in extreme periphery and tears of retina. Contusio bulbi. Direct hit.
51	C. N—	25	Civilian	—	L.E. struck by racket ball	R.E. $\frac{6}{6}$; L.E. $\frac{6}{12}$	Rupture of choroid close to macula. Contusio bulbi. Direct hit.
52	E. C—	23	Private	11th Royal Warwicks	Date ? ; Loos ; Minnen Werfer exploded 6 yds. away	R.E. $\frac{6}{24}$; L.E. enucleated	R.E. : Two small corneal scars. Fine pigmentary changes all over the fundus, with some hæmorrhage in extreme lower periphery, organising. Force of explosion spun him "round and round like a top." Injury to R.E. due to "windage" or indirect from left side.
53	R. C—	?	Private	1st Seaforths	— 8 : '15 ; La Bassee ; explosion, struck by sandbag	R.E., L.M.O. ; L.E. $\frac{6}{6}$	R.E. : Solid detachment of retina due to choroidal hæmorrhage. Has scar ¼ in. square above, in right eyelid.
54	S. H. R—	42	Private	76th Canadians	1 : 11 : '16 ; Somme front ; shrapnel	R.E. enucleated at Boulogne ; L.E. $\frac{6}{12}$	Shrapnel pierced root of nose after entering right orbit. Scattered pigmentary disturbance on inner side of fundus. Disc normal. No white areas seen.
55	D. B—	20	Private	2nd Lanc. Fusiliers	1 : 7 : — ; Somme ; splinter of bursting shell	R.E. blind ; L.E. P.L.	R.E. shrunken. L.E. : Blood-staining of cornea following hæmorrhage, result of iridectomy for blocked pupil.

No.	Initials.	Age.	Rank	Corps	When, where, and how wounded.	Vision of each eye.	Condition of eye and notes in the text.
56	L. W—	25	Lieut-enant	—	10 : 1 : '17; bursting shell	R.E. enucleated; L.E. $\frac{5}{12}$	L.E.: "Vossius" concussion ring, typical. Sub-hyaloid hæmorrhage in lower periphery. Lens changes disappeared early in April.
57	G. C—	44	Bom-bardier	R. G. A.	27 : 12 : '16; Arras; bombardment; "windage"	R.E. $\frac{6}{6}$; L.E. $\frac{6}{24}$	L.E.: Floating vitreous opacities. Curtain of opacity in periphery below, and in midline a mass of red coagulated blood. No rupture of choroid. He was 200 yards away from bursting shells. Bombardment lasted from 10 p.m. to 3 a.m.; he then slept, and on waking noticed mist in eye; concussion or coincidence?
58	B—	29	Captain	1/4 Royal Berks.	August, 1916; bullet; "through and through" behind malar bone	R.E. $\bar{c} - 8 = \frac{6}{60}$; L.E. $\bar{c} - 8 = \frac{6}{60}$	Pt. highly myopic. Numerous choroidal ruptures seen in both eyes with hæmorrhage. Has a transparent detachment of retina in the L.E. up and in.
59	R—	24	Lance-Corporal	7th South Staffs.	27 : 12 : '16; Ancre; "whiz-bang" exploded 10 yd. away; "windage"	R.E. $\frac{6}{60}$; L.E. $\frac{6}{6}$	Both eyes showed extensive scattered pigmentary changes of retina without any other grosser lesion. The changes in R.E. were central; in L.E. peripheral and lens marked. "Windage."
60	B—	30	Lieut-enant	4th Royal Sussex	29 : 12 : '16; Riviere; shell exploded in front of dug-out as he was leaving; "windage"	R.E. $\frac{6}{12}$; L.E. $\frac{6}{6}$	Right optic disc decidedly paler than left. Slight disturbance of pigment downwards and onwards. Has lost colour vision. Field limited generally. No central scotoma, but general haze.

61	C—	22	2nd Lieutenant	R. F. A.	22 : 1 : '17 ; Marieux ; aeroplane accident; pilot on machine which fell 300 ft. but side-slipped at the last	R.E. $\frac{6}{6}$; L.E. $\frac{6}{18}$; later $\frac{6}{6}$	L.E. had a central scotoma 10 in. in extent. Machine fell and he struck his nose violently, and also the back of his head. Probably had hæmorrhage into base of his skull. Vision of L.E. cleared up entirely.
62	S—	25	—	4th Corps 1st Lanc. Fusiliers	4 : 12 : '16 ; Guillemont ; bursting shell	R.E. $\frac{6}{60}$; L.E., fingers 6 ft.	"Windage." Was knocked over by exploding bomb. R.E.: Rupture of retina. L.E.: Rupture of iris. Lymph on anterior surface of lens. Extensive rupture of choroid in macula. Has some scars on face.
63	A. W. C—	22	Lieutenant	7th Leinsters	2 : 2 : '17 ; Plug Street ; trench mortar	R.E. $\frac{6}{24}$; L.E. $\frac{6}{6}$	"Irido-dialysis." R.E.: Star-shaped opacity at post centre of lens. Concussion cataract. Fundus healthy. Direct hit on eye.
64	R. W—	48	Private	7th Leinsters	26 : 1 : '17 ; Somme ; "something exploded in the incinerator" and struck his eye	R.E. L.M.O ; L.E. $\frac{6}{6}$	R.E.: Lens dislocated. Concussion cataract at posterior pole. Vacuolation. Iris torn and pupil dilated. Blood in anterior chamber. Direct hit on eye.
65	G. S—	20	—	Argyle and Sutherland Highlanders	26 : 3 : '16 ; Arras ; bullet	Blind ; no P.L.	"Through and through" wound. R.E.: Optic nerve severed. Rupture of choroid. L.E. shrunken. Posterior rupture of globe. Enophthalmus.
66	R. L—	26	Private	King's Own Scottish Borderers	9 : 7 : '16 ; Loos ; shell explosion	R.E. blind ; L.E., P.L.	R.E.: Lens opaque. F.B. in eye. Eye subsequently lost by septic irido-cyclitis. L.E.: Large solid detachment of retina due to blood in choroid.
67	P. H. E—	41	Lieut.-Comdr.	R.N.V.R.	13 : 11 : '16 ; Somme ; bullet wound	R.E. $\frac{6}{6}$; L.E. blind	"Through and through." Left optic disc severed. Rupture of choroid and retina near the disc. Altitudinal squint. L.E. lower than right. No movement upwards.

No.	Initials.	Age.	Rank.	Corps.	When, where, and how wounded.	Vision of each eye.	Condition of eye and notes in the text.
68	J. A—	23	Private	Royal Irish Fusiliers	25 : 4 : '15 ; France ; shrapnel	R.E. blind ; L.E., shadows	Extensive rupture of choroid and retina. B.E. remains of posterior cortical cataract. A group of oval vacuoles irregularly arranged, about 20 in number.
69	S. H. H—	28	Private	12th London	20 : 2 : '17 ; Neuve Chapelle ; mass of hard earth	R.E. $\frac{6}{6}$; glasses $\frac{6}{6}$; L.E. blind	L.E. : Striate keratitis. Rucking of Descemet's membrane T. — 2. Contusio bulbi. Lens dislocated. Post-cortical stellate concussion cataract. Sphincter pupillæ ruptured.
70	R. W. S. S—	27	Private	1st Coldstreams	May, 1915 ; Neuve Chapelle ; bullet	R.E. blind ; L.E. enucleated	"Through and through" wound. R.E. : Optic nerve severed. Pupil widely dilated and fixed. Remains of posterior cortical cataract. A group of oval vacuoles, looking like air-bubbles, on posterior surface of lens.
71	C. H. R—	23	Lieutenant	1st Middlesex	1 : 7 : '16 ; Somme ; bullet	R.E., bare P.L. ; L.E., no P.L.	"Through and through" wound. Rupture of choroid and retina and optic nerve on left side. Both pupils displayed hippus when examined January and March, 1917. Slow rhythmic contractions and dilation.
72	E. McA—	29	Rifleman	Royal Irish Rifles	1 : 3 : '17 ; Bouchavesnes ; shrapnel	R.E., L.M.O. ; L.E. $\frac{6}{6}$	Irido-dialysis T. — $\frac{1}{2}$. Posterior cortical concussion cataract, stellate in form. Vacuolations. Anterior part of lens also turning opaque. Eye removed subsequently on account of pain.
73	S. B—	24	Private	East Kent Yeomanry	6 : 9 : '16 ; Somme ; bullet	R.E., *nil* ; L.E. $\frac{6}{60}$	R.E. enucleated. L.E. : Large coarse mass of pigment at macula, with fine changes peppered over large area of fundus. No evidence of rupture of choroid or retina.
74	T. N—	70	Civilian	—	13 : 3 : '17 ; struck by mass of soft clay thrown by fellow-workman	R.E. $\frac{6}{6}$; L.E. $\frac{6}{24}$	In front of nodal point of left lens is a circular discoid cataract, which can be seen both by transmitted and by focal illumination. It was distinctly deep to anterior capsule ; it was similar in size to the "Vossius" concussion ring, but some fine opaque dots filled the interior of the ring.

75	W. W—	26	Private	7th Leicesters	26 : 2 : '17 ; Vermelles ; shrapnel	R.E., L.M.O. ; L.E. $\frac{6}{6}$	F.B. removed by magnet. at Boulogne. Irido-dialysis. Post-cortical concussion cataract. Stellate. Some deposit on anterior surface of lens ; also palisade arrangement of vacuoles at lens periphery.
76	D. T. M. S—	30	Lieutenant	Royal Welsh	24 : 9 : '15 ; Festubert ; bullet	R.E. $\frac{3}{60}$; L.E., *nil*	"Through and through." R.E. detachment of retina, result of hæmorrhage. Has four air bubbles on posterior surface of lens. Vacuoles. L.E. : Optic nerve severed.
77	H. I. B—	30	Private	Rifle Brigade	6 : 2 : '17 ; Combles ; explosion of cartridge	R.E. $\frac{6}{6}$; L.E., no P.L.	Anteversion of torn iris. Posterior surface of iris seen from the front, upper half of iris torn. Irido-dialysis. T. — 1. Lens dislocated. Case seen by courtesy of Mr. Dawnay at Great Northern Hospital.
78	B—	31	Private	2/5 Leicesters	16 : 3 : '17 ; Favricourt ; "whiz-bang"	R.E., P.L. ; L.E. $\frac{6}{9}$	Was rendered unconscious for an hour by explosion. Has two small scars in upper lid of R.E. Anterior capsular opacity, square-shaped. On magnification opacity shows minute dark points making up opacity and spreading beyond it. Folds in capsule can be seen distinctly. Iris ruptured. Lens dislocated. Vitreous opacity. No F.B. in eye.
79	G. S—	26	Private	R.A.M.C.	25 : 12 : '16 ; Albert ; shell-burst	R.E., fingers 3 yd. ; L.E. $\bar{c} - \frac{8}{15} = \frac{6}{12}$	Large shell burst 12 ft. away. Twelve hours later headache. Eight days after noticed sight begin to fail. Billowy opaque detachment of retina below. Floating vitreous opacities. Minute superficial scar on cornea.
80	G. L—	41	Civilian	—	12 : 3 : '17 ; Northfleet ; piece of wood struck L.E.	R.E. $\frac{6}{5}$; L.E. $\frac{6}{30}$, indirectly	Circular hæmorrhage at macula deep to retinal vessels. When this absorbed an area of choroid-retinal change could be seen.

Opening Paper.

Concussion injuries of the visual apparatus in warfare, of central origin.

By S. A. Kinnier Wilson.

It is imperative at the outset to indicate as precisely as possible what type of case is to be assigned to the above category, for the whole subject of concussion and commotio is still surrounded with considerable obscurity, and looseness of conception is responsible for a certain amount of clinical confusion.

The difficulty is not lessened by the fact that the usual definitions of concussion and commotio do not command universal consent. Cerebral concussion is commonly understood to signify a violent shaking or jarring of the cranial contents without any structural lesions, and it seems certain that in some cases which have proved fatal the most painstaking investigation has failed to reveal any recognisable anatomical lesion. Yet the idea of a *morbus sine materie* is so repelling that to explain such cases a condition of "shock" is postulated, *i. e.* a reflex inhibition of certain cerebral mechanisms essential to life, or a condition of commotio, *i. e.* molecular concussion, producing the same result. Not a few writers, however—Kocher (1), Page (2), Thorburn (3), Stolper (4), Schmaus (5), Hartmann (6), and others—deny the reality of spinal or cerebral molecular concussion (commotio), maintaining, on the contrary, the impossibility of a severe or fatal cerebral or spinal affection without any definite anatomical lesion.

In most cases of severe blows on the head, as a fact, which have been diagnosed as concussion and which have proved fatal, examination has shown the presence of capillary hæmorrhages, minute lacerations of cerebral substance, and consequent disintegration of myelin, such

as are sufficient to account for the clinical symptoms. Further, as Warrington (7) has remarked, and as any who have considered the subject must have realised, there is, with the blow, a sudden violent dispersion of cerebro-spinal fluid, which tends to escape too abruptly by the natural exits. Hence ensues a tearing of lymph channels and injury to the adjacent cerebral tissues.

In non-fatal cases where the symptoms, though definite enough, have eventually cleared up completely, it is justifiable to assume organic lesions such as have been enumerated, which have not, however, been sufficiently widespread or serious to effect permanent impairment of cerebral function. For such cerebral conditions, where one has reason to suppose recognisable, if temporary, organic changes have occurred, it is preferable to employ the term cerebral contusion.

For the purposes of this paper, then, cerebral contusion may be taken to be the pathological basis of the condition known clinically as cerebral concussion.

Now it does not appear practicable to draw a hard-and-fast line between such cases and those, being slighter in degree, to which the term commotio, *i. e.* molecular concussion, may be applied. A knock on the "funny bone" produces a transient disturbance of sensibility in the ulnar distribution, as a basis for which we may perhaps postulate transient oscillations in the axoplasm of the nerve fibrils; whatever it be we must imagine some change taking place. When the pressure of one limb on the popliteal nerves of the other results in the foot's becoming "asleep," we suppose a disturbance of function whose basis may be interruption of periaxonal or perineuronal lymph circulation. Let the pressure continue longer, as in, say, a crutch palsy, and we have an actual organic impairment of function which may take weeks to clear up. We call such a condition, *faute de mieux*, a pressure-"neuritis," but the accuracy of the term, pathologically speaking, is problematical; indeed, whether any recognisable change could be determined microscopically is questionable. It is

impossible to say in such a series of instances at what precise stage the term "structural change" may be fairly adopted, and the difficulty is not less in regard to cerebral commotio. Oppenheim (8), in opposition to the other authorities quoted above, is unwilling to relinquish the conception of molecular concussion or commotio, although he rather admits that for him the conception does not exclude the possibility of such fine organic changes as alteration in capillary or lymph channel walls, disintegration of the myelin in individnal fibres, and so on. From the nature of the case, in the present state of knowledge, the matter must remain in the realm of hypothesis.

While, then, the question of the actuality or otherwise of molecular concussion or commotio is still *sub judice,* and to some may be of mere academic interest, it should not be overlooked that from the clinical side it presents features of importance. In the course of the war there have come under my notice a large number of concussion lesions of the spinal cord, of every kind, which may range from complete pulping of the cord at a given level, with dura mater intact and untouched, to a concussion such as reveals itself solely by transient symptoms of the type unfortunately termed "functional." Clinically there is, at the one extreme, a total motor and sensory paraplegia, at the other, the tremulous weakness, paræsthesiæ, and exaggerated reflectivity of "functional" diseases. For practical purposes it is convenient and serviceable to divide cases into organic and "functional," but more than one case has came under my notice that has clearly been a sort of "missing link" between the two groups (1). The evidence undoubtedly suggests that in certain cases so-called "functional" symptoms, following immediately on spinal concussion, are but the expression of an organic alteration minimal in degree and insufficient to result in the appearance of organic symptoms as ordinarily known. The significance of this will be seen subsequently in regard to the visual apparatus.

[1] In this connection some cases reported by Elliott (9) are of interest.

Experimental work on the subject of concussion and commotio has been attempted to some extent, but it does not help to clarify our ideas as much as might have been expected. Schmaus (5) has demonstrated, in artificial concussion of the cord in kittens, swelling and degeneration of axones and fragmentation of myelin, without any gross pathological lesion. It has been shown that a blow upon the head of an animal may occasion this myelinic change in the fibres of the medulla, and even of the cord, presumably by dispersion of the cerebro-spinal fluid. Much the best and most scientific experimentation has been conducted by Alan Newton (10), in the laboratory of the late Sir Victor Horsley. Newton allowed a glass rod, 8 mm. in diameter and weighing 50 grm., to fall upon the exposed spinal cord from varying heights, accuracy of direction being obtained by means of a glass tube, 1 cm. in diameter, through which the weight fell. So marked was the effect from heights of 40, 20, or 10 cm. that the height was reduced to 2 and finally to only 1 cm. The latter was a feeble blow, producing no visible effect on the cord, yet it completely abolished motor conduction for several days, both in the cat and the monkey. It is important further to note that a dissociation in conductivity was obtained, sensory conduction being still demonstrable though motor inhibition was complete. The anatomical changes found in such animals were described by Newton as "trivial"; "they consisted of small hæmorrhages in the grey matter, usually in the basal region of the posterior cornua or in the posterior grey commissure, scattered swellings of axis cylinders, and disintegration of myelin sheaths."

These experiments suggest that there is an organic basis for the transient disturbances of function seen in so-called "railway spine" and allied conditions of traumatic neurasthenia, and they bear out what has already been remarked, that "functional" symptoms may be the expression of minimal organic changes. They also prove the occurrence of a relatively surprising amount of tissue change from a feeble impact on the exposed cord, and show that the

blow capable of producing merely a molecular concussion must indeed be slight.

When, from these necessary preliminary considerations, we pass to the question of concussion injuries of the visual apparatus in warfare, of central origin, we are compelled to the conclusion that a molecular concussion, or commotio, is almost certain to be of great rarity, for the simple reason that the impacts of high-velocity projectiles, or even of shell fragments or spent bullets, are tremendous, to a degree far beyond that of the glass rod utilised in Newton's experiments. The Shooting Editor of the *Field* has written a letter on the subject, which is quoted by Musson (11) in his recent paper on the finer histological changes in the spinal cord following bullet wounds of the cord substance or shock to the vertebral column, and which contains useful information on the topic under discussion. It relates to the ·303 Mark VII service bullet, weighing 174 gr. "Assuming that such a bullet strikes the body at 200 yards with 1000 foot-lb. energy, and is pulled up in 3 in. by the skin, muscles, and spinal column, the resistance offered by the tissues is on the average . . . 4000 lb. This 4000 lb., being the resistance offered by the tissues, may be regarded as the static pressure of the bullet acting against the tissues." That an impact so tremendous should render the occurrence of a mere commotio practically an impossibility is not in any way surprising. And even though a bullet be spent, or a shell fragment be pulled up by a steel helmet, the force of the blow must still be much beyond that which has often produced a concussion or commotio in civilian cases.

It will be seen, therefore, at once, that while gross contusion lesions of the central visual apparatus are common enough, one may have to seek far and wide ere one can find a case which, for the purposes of this communication, may be admitted into the category of visual concussion, where one may suppose the organic disturbances are of the trivial nature that has already been described.

At Fulham Military Hospital and Napsbury War Hospital a large number of head injuries of all sorts have been referred to me; at the National Hospital and at Lord Knutsford's Special Hospital for Officers, in addition to my own cases, I have had the opportunity, through the kindness of my colleagues, of going over a very considerable number of head cases with visual symptoms, and it may be said at once that only one case, described below, approaches the conception of an uncomplicated visual concussion, and even it is by no means free from objections. Two other cases to be quoted are instances of visual symptoms following blows on the head, but the mechanism of production of these symptoms is not that of concussion, and they are cited by way of contrast. After two and a half years one is still looking for a case where a direct blow on the head from any form of projectile, without gross wounding, has resulted in an uncomplicated visual concussion or commotio analogous to the cases of spinal cord or peripheral nerve concussion or commotio that have been, relatively speaking, common.

Let it be observed that all cases of visual disturbances from shell explosions *in the vicinity of* the individual concerned are purposely excluded. They belong to another group. Such are common and well known (see, for instance, Myers's (12) cases). I am not concerned to deny that violent dispersion of the air is a physical factor which may produce physical, *i. e.* organic, injury; on the contrary, two genuine cases of organic lesion of the spinal cord from shell explosions, without any outward mark, have come under my notice, and others (*e. g.* Ravaut (13)) have described similar examples. As long ago as the battle of Trafalgar, as Mahan tells, Nelson's clerk was "killed by the wind of a ball, though his person was untouched." Still more may such dispersion occasion transient disturbances of the kind we call "functional," though they may have some subtle organic basis underlying them. But, obviously, other factors, of a different class from those leading to commotio, come into

play in such cases—"shell-shock" is a complex resulting from several agencies. Further, it is difficult to imagine how visual impairment from "shell-shock" could be of central origin, unless, of course, the patient, knocked over by a shell explosion, were to strike his head in falling. The peripheral visual apparatus is exposed, whereas the central apparatus is protected by the cranial box; it is the peripheral mechanism that must take the brunt of such "windage" effects. A consideration of the frequently accompanying phenomena of photophobia, blepharospasm, of "eyes burning," and so on, bears this out. To attempt to demonstrate the possibility of central defects by the action of diaschisis from peripheral concussion would be impracticable. Unless an exact history of a fall on the head from a shell explosion were forthcoming, the occurrence of central concussion from "shell-shock" can scarcely be established. Personally, no such case has come under my observation out of a very large number of cases of "shell-shock."

Secondly, all cases of head wounds with resulting more or less permanent organic visual defects are excluded. Many cases of ordinary head injuries in the occipital region, with resulting scotomata and hemianopias of various kinds, have come under my notice. Only a relatively gross organic defect in the form of loss of cortical substance, cortical or subcortical hæmorrhage, etc., will explain the condition, clinically, so that they do not belong to the concussion group. It is a matter of common observation that in the majority of such cases a period of practically complete blindness is the initial phase, much of which subsequently clears up. In Marie and Chatelin's recent paper (14), for instance, it is noted in passing that most of their patients with varieties of hemianopic defect were blind for so many hours or days at the outset. Where, however, we have actual gross injury to the skull and intracranial contents, it is impossible to determine whether cortical laceration, or œdema, or subdural or subarachnoid effusion, or capillary oozing,

or other obvious organic change, or merely a concussion or commotio, is the underlying factor for such symptoms as clear up completely. For this reason, if for no other, one cannot take severe head injuries into the class which forms the subject of this communication.

We are left, then, with cases where a blow on the head produces visual symptoms that clear up, but does not result in a permanent hemianopia in any of its varieties or other type of organic symptom. Though the case about to be quoted admittedly does not come up to the proposed criterion, it is the one least open to objection.

Case 1.—Pte. A. B—, æt. 19 years, 7th Shropshire Light Infantry, with a negative previous and family history, was wounded at 4 p.m. on December 14th, 1916. At the time he was stooping down, cutting wood, in a little dug-out. He was hit on the back of the head, in the mid-line, just below the margin of his steel helmet, by a fragment of shell, and lost consciousness for, at the most, three minutes. He came to, to find a mate moving his right arm, which had also been hit and which was very painful; it was, perhaps, this pain which reached his sensorium and brought him round. At the same time he had racking pains all over the head, was very sick, and found he was completely blind in both eyes. He kept asking whether his eyes had been hit, as he could see nothing at all, only blackness everywhere. He remained thus blind for between forty and forty-eight hours. On the third day he found that he was beginning to see dimly with both eyes, and trying each separately he found he could see a little with each. He saw a hand holding a medicine glass, but nothing beyond that; such objects appeared very near him, but not altered in size; there was, in other words, no dysmetropsia with this defect in the appreciation of distance. The same afternoon he was operated on and a small piece of loose fractured bone removed from the head wound; again, two days later, a second small splinter was taken out. Towards the end of the first week, when his sight was improving, he frequently

noticed a succession of faint, dark, shadowy forms and figures moving to and fro in front of his eyes, apparently. He never could make out quite what they were, except that they were always shadowy and dark, with no colour in them. Sometimes they were still. He used to shut his eyes to get rid of them, but this procedure did not banish them, or, rather, sometimes it did and sometimes it did not. These subjective visual phenomena disappeared after about a fortnight. Of them the patient remarked that they always appeared smaller than reality, whereas objects seen in front of him were of normal size. Only

Fig. 8.

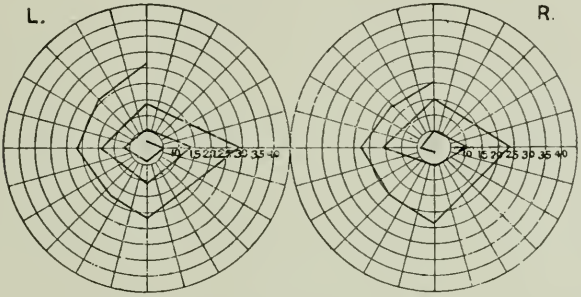
Fields for White in Case 1 (April, 1917).

the hallucinations were microscopic. It should be stated that at no time were the patient's eyes bandaged, nor was there at any time either photophobia or blepharospasm.

Very slow improvement took place, and when the patient came under my observation two months later, his condition was as follows: The pupillary reactions were normal. The optic discs were normal, with no indication of previous neuritis. There was no trace of any form of hemianopia, or of scotoma, central or otherwise. Vision was: R., $\frac{6}{60}$; L., $\frac{6}{60}$; Jaeger, 14 R. and L. There was no headache, giddiness, or sickness. Careful routine examination of the nervous system revealed no abnormality except in regard to the visual fields. Here there was a concentric diminution of both fields, not quite symmetrical,

both for white and colours, and also a marked condition of visual fatigue, so that excellent examples of a helicoid curve were obtained in both eyes. This helicoid was present both for white and colours, and was rather flattened

Fig. 9.

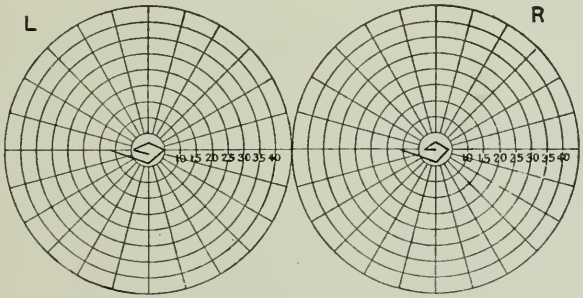


Spiral Fields for White in Case 1 (May 4th).

in a vertical direction. Bjerrum's screen was always used in testing.

The patient was re-examined at intervals during the

Fig. 10.



Spiral Fields for Red in Case 1 (May 4th).

next two months, the improvement continuing slowly. On May 4th his vision was: R., $\frac{6}{20}$; L., $\frac{6}{15}$; Jaeger, R., 6; L., 4. The fields were distinctly better than before in the sense of being less constricted; the helicoids were again obtained. (Figs. 8, 9, and 10.)

The patient is still under observation.

As the X-ray photograph shows, and the fact of operation indicates, we are dealing in this case with a lesion of undoubted severity, the outer table being splintered and the inner being cracked, and perhaps slightly splintered too. It is not to be denied that with such a lesion submeningeal effusion, or intracortical capillary hæmorrhage, or tearing of lymph channels, or myelin fragmentation are clearly possibilities to be envisaged. But, on the other hand, it must be borne in mind that the patient was unconscious for, at the most, three minutes, that there has been no evidence of intracranial pressure, that the absence of any form of scotoma or hemianopia is proof of the general integrity of the visual cortex, and that the visuopsychic areas are similarly intact.

If, therefore, in the ordinary sense, there is structural integrity of the visual cortex, we are justified in associating the visual phenomena with the fact of a violent concussion over the occipital poles.

It is not forgotten that when bullets are directed against water-containing mediums, as the Shooting Editor of the *Field* states, they set up a hydraulic force which acts radially through the mass. The brain, however, does not respond exactly as a water-containing medium, and the reaction to a blow is not uniformally distributed throughout its mass; as Warrington says, it is most intense at the point of application, and in those parts of the brain opposed to fixed points—the points of contre-coup. Now, in the case under consideration there is no clinical evidence to suggest a condition of contre-coup, hence the conclusion above stated appears to me inevitable.

An objection of another kind must next be considered. It may be said this is merely a banal case of traumatic hysteria, as helicoid and concentric diminution of the visual fields were described long ago in that neurosis, and have been supposed to be pathognomonic of it. (By the way, it may be worth remarking that the existence of helicoid fields in hysteria is one of those typical state-

ments which are copied from one text-book to another in uncritical fashion, and which may be found to have little satisfactory clinical support. None of the more modern works on hysteria contain any fresh clinical instance of the condition, while some authorities do not even refer to it. It is, in short, a matter which calls for reinvestigation.) But against this contention several arguments may be raised.

(1) With the exception of the visual symptoms no impairment of function of the nervous system was discovered. Were the case, then, one of hysteria, it must fall into the category of monosymptomatic cases, which in ordinary experience are rare, but do nevertheless occur. Now, concentric diminution of the visual fields has never been described as an isolated phenomenon in hysteria. Personally, I have never seen an instance of this visual defect in hysteria unaccompanied by cutaneous anæsthesia. This is in agreement with the findings of Voss (15) in his recent monograph, and Féré (16), Wilbrand and Saenger (17) and others have also stated that concentric diminution of the fields is found only in cases with some form of anæsthesia elsewhere. In face of this opinion the contention that the case is hysterical is seriously weakened.

(2) The war has afforded proof, over and over again, of the fact that an interval of hours or days intervenes between the shock-producing stimulus and the development of a traumatic psychoneurosis, such as hysteria. The French say "there is no hysteria in the firing line"; time must elapse, though it be measured but by hours, for subconscious complexes to be formed—"une période de méditation" is necessary, a subconscious rumination. Instance after instance of the action of this mechanism has come under my notice; it is, in fact, almost a commonplace. Now, in the case under consideration the patient was struck on the back of the head, was unconscious for three minutes, and regained consciousness to find himself completely blind. In forty-eight hours he was beginning to

improve. Being ignorant of the details of cerebral anatomy, he thought his eyes had been hit. In view of these circumstances it is suggested that to attribute his symptoms to post-traumatic auto-suggestion or other subconscious activity is unjustifiable and erroneous.

That traumatic neurasthenic symptoms may immediately succeed a shell explosion or a direct injury, such as being buried, is recognised by all who have been working on the neurological side of the injuries of warfare, but a distinction must be drawn between neurasthenic manifestations and hysterical manifestations. Restriction of the visual fields and helicoid curves, without being in the strict sense pathognomonic, are characteristic of the latter condition.

Instead, therefore, of regarding the case as one of traumatic hysteria of the monosymptomatic variety, I hold that we are dealing with a true cerebral concussion, or commotio, affecting the visual system, and some at least of its value consists in the light which it throws on the mechanism of production of so-called "functional" phenomena. That is to say, so far from hysteria mimicking organic disease in this case—a process which Charcot and his pupils maintained was of common occurrence—we have here an instance of organic disease—for concussion is an organic state—mimicking hysteria, where the symptoms produced by a violent blow, without presumably any structural change, simulate, though they are not identical with, those produced by subconscious activity.

A generation ago Hughlings Jackson asked what are the symptoms of "functional" disease of the cerebellum, and the question remains unanswered. Of the "functional" diseases of other intracranial mechanisms we are equally ignorant. It may be that to put the matter in this light will evoke the response that all symptoms are "disturbances of function," and that to try to distinguish between "functional disturbances" and "disturbances of function" is a mere quibble. So much the better. I believe this case may be taken to suggest that in certain cases the outward manifestations of a minimal organic

disturbance are of the type hitherto supposed to be characteristic of "functional" disease. It becomes matter for discussion whether the "shock" of a concussion may not affect a change in cortical bio-chemical activity, the expression of which is comparable to the "functional" disturbance called hysteria.

By way of contrast, a case may be selected where, notwithstanding head injury and cerebral concussion amounting perhaps to contusion, the visual symptoms are, in my opinion, derived from another source.

Case 2.—Sapper C. D—, æt. 44 years, was wounded at Neuve Chapelle in March, 1915. When walking down a path from the village to the trenches he was struck by fragments from a shell which exploded in his immediate vicinity, and fell unconscious. He does not remember anything till his arrival in hospital, two days later, and it is not known whether he remained unconscious all that time. He was wounded in the forehead above the right eye, the frontal bone being splintered, and above the right ear, where apparently the bone remained intact; there was a cut on the left upper eyelid and dirt in the left eye. When he came to he found the left eye bandaged, while the right was free. The bandage remained on for some ten days, and when it was permanently taken off he discovered that he could not see with the left eye, while the right was normal. With the left eye everything was "a blur of golden light," "like a sun with its rays pointing out in all directions," so that he could distinguish nothing. The visual condition remained unchanged till six months ago, when he noticed he could see out of a small area in the upper and outer field on the left side. This has not apparently enlarged during these six months.

On examination, in March, 1917, the actual condition of the left eye was seen to be normal in all respects. The pupil reactions were normal on the two sides. The optic discs were normal and identical in colour. The double prism and "friend" tests unfortunately both proved unsatisfactory, mainly because they had been tried before

and because the patient's answers were non-committal and obviously not frank. However, he was found on further examination to be "deaf" in the left ear and anæsthetic to pin-prick over the left half of the body and limbs, including the left half of the tongue.

In this case, conceivably, we are dealing with a concussion effect on the left peripheral ocular mechanism, but in view of the other symptoms it is to be placed in the category of traumatic hysteria. Notwithstanding the genuine cerebral concussion produced by the head injuries, the mechanism of development of the blindness is of a different kind altogether from that of the first case.

Another case may be selected.

Case 3.—Pte. E. F—, æt. 45 years, 1st Suffolks, was sniping from the trenches at Zonnebeke one evening in September, 1915, when he was hit on the left side of the head and face by fragments of what he maintains was an explosive bullet, which struck some object only a few inches away from where he was standing. He fell unconscious, and remained in that state for about one hour. On coming round he discovered he was blind in both eyes, and remembers that he noted this at a time when the bandages which covered both eyes were temporarily removed. On about the tenth day after the wound the bandage was permanently taken off the right eye, and he found vision with that eye was normal again. The left eye remained covered with a shade for some more weeks, and when it too was removed he declared he was quite blind in the left eye still, and he has remained so ever since.

On examination in March, 1917, the left eye was seen to be normal in all respects, with normal pupil reactions. The optic disc was normal and identical in appearance with the right. No defect of any kind was discovered in the nervous system, either organic or "functional." With the double prism and the "friend" tests the patient was completely caught out, and the nature of his visual condition at once demonstrated.

Here, again, in spite of the general cerebral concussion, unconsciousness, and immediate blindness, the persisting visual defect is clearly seen to be of the nature of so-called "subconscious malingering." Whether the blindness noted at the outset was a concussion phenomenon is questionable, in view of the facts that the patient was not struck on the back of the head, and also that he knew he had been hit in the face and round the left eye, was told there was dirt and sand in both eyes, and remained with both bandaged for some ten days. To exclude the possibility of a peripheral concussion would be more difficult, and it is conceivable we have here a hysterical symptom erected on an existing slight organic basis. The case is quoted to illustrate this difficulty in determining whether a persisting symptom is an original symptom, as well as the further difficulty of assigning relative importance to the more or less synchronous actions of more than one ætiological factor.

Some considerations arising out of the subject deserve a little attention.

It may be taken as axiomatic that every case of head injury from gun-shot wounds of any kind in warfare must be associated with cerebral concussion, whether it be a cerebral contusion or something considerably less in degree. The immediate effects of concussion are generalised, in the shape of, *inter alia*, unconsciousness. The sequelæ may be localised, or generalised, or both. Among the latter are headache, asthenia of cerebral type, not infrequently "cerebral irritation," as revealed by profound restlessness, inattention, and irritability. More interesting for our purpose are localised sequelæ. Some special function of the brain may be permanently lost or impaired, *e. g.* a localised retrograde amnesia has not seldom been observed. Montier (18) has recently published a record of his investigations of labyrinthine impairment of function after cerebral concussion from head injuries in warfare. In a series of 46 cases of gun-shot wounds of the head

no fewer than 40, *i. e.* 87 per cent., showed evidence of labyrinthine concussion or defect of function on objective examination, independently of the site of the injury. That so high a proportion is reached can be understood from the situation of the labyrinth, embedded in the bony framework of the cranial box. This raises the question of the relative vulnerability to concussion of the special sense systems of the cerebrum. I have no data for taste and smell, but, as far as the others are concerned, in my experience visual impairment after generalised cerebral concussion is much rarer than labyrinthine impairment, and certainly rarer than auditory impairment. It is interesting to note in this connection that Mott (19) finds in his experience of "shell-shock" cases that defects of vision are relatively uncommon as compared with deafness and hyperacusis. Probably the anatomical situation of the central visual apparatus acts as a protection, while for the same reason as the labyrinth the peripheral auditory mechanism is prone to be concussed wherever the actual head injury be.

The question of the interpretation of the visual phenomena in the first case quoted is of considerable importance. The view here put forward is that certain symptoms usually held to be "functional" in type may be the expression of a direct visual concussion, undoubtedly an organic condition. These symptoms are concentric restriction of the visual fields and the occurrence of helicoid or spiral fields with certain tests.

(1) In regard to the first of these, it must be borne in mind that such concentric diminution, supposed to be characteristic of hysteria, does certainly occur from head injuries. In the series recorded by Marie and Chatelin (14) there are two cases where the visual fields are almost identical with those obtained at the first examination in my case. In these two, however, "il s'agissait de traumatisme grave de la région occipitale atteignant les deux sphères visuelles." In many other cases, as has been already remarked, an occipital head

injury is ushered in by general blindness, for some days or hours: "le syndrome de la cécité corticale relevait chèz ces blessés de commotion ou de compression de tout le territoire visuel cortical, non de déstruction étendue comme dans les deux cas." Marie and Chatelin therefore regard the general blindness as due to compression or commotio, whereas, by implication, the constricted fields of their two cases have a different origin. In each case the patient was blind for many months, and macular vision was the first to return. Whatever be the explanation—and the authors do not venture on one—the important point is that such fields are clinically indistinguishable, as far as constriction is concerned, from those of hysteria. There is no mention of subconscious vision over the rest of the field, and it may confidently be assumed that the two patients showed none of the usual phenomena of hysteria.

Again, in the series published by Lister and Holmes (20) there is one (their Case 20) where the patient was shot through the head from side to side, above and behind the pinnæ. "Six weeks after the infliction of the wound he could only read large letters, and his visual fields were limited to a zone of about 10° around the fixation point. He was able to recognise colours by macular vision." There is no mention or suggestion of hysterical symptoms, and, of course, the organic nature of the lesion is undoubted, it being fairly certain that the optic radiations were injured. The authors state that in their opinion "the interpretation of his visual disturbance is doubtful." Here, once more, is an organic case with a visual defect of constricted fields, as in hysteria.

My case is a fourth, therefore, where a moderately severe organic lesion in the occipital region is followed first by general blindness, and subsequently by concentric diminution of the fields, in a marked degree, and, like these others, with no trace whatever of any symptom of the hysterical psychoneurosis. It differs from Marie and Chatelin's cases in the short duration of the blindness; in fact, it is a

much milder case as far as general symptoms are concerned. Lister and Holmes say that "telescopic vision" is "not uncommon in patients who have been shot through the head in the lower parietal or temporal regions," and the above case is quoted by way of illustration. They do not, however, make any suggestion as to the pathogenesis of the condition in these cases.

By way of contrast, in regard to this one point, a case recorded by Eisdell Moore (21) may be quoted. The patient was shot through the back of the head from side to side, and was at once totally blind. On the seventh day he could distinguish "large objects"; on the tenth he could count fingers (distance not specified); on the fifteenth "there was some limitation of the right field of vision." Within three weeks his fields of vision were "normal." Unfortunately perimetric examination was not practicable. The author's comment is: "From the rapidity of recovery it is evident that blindness was due mainly to concussion, yet it seems extraordinary that a bullet could pass through both occipital lobes without causing some permanent interference with vision." It is possible, however, that a bullet might pass through the occipital lobes without injuring either the optic radiations or the area striata. Notwithstanding the incomplete record this is a case of direct occipital jarring without notably constricted fields, and, without doubt, most workers on the subject have seen analogous cases.

From what has been said, then, it must be conceded that in certain cases of organic injury of the central visual mechanism by concussion or otherwise the result is constriction of both fields, occurring by itself, and without evidence of subconscious vision in the remainder.

(2) I have not been able to find any reference to helicoid fields in recent war literature, with the exception of a comment by the late Mr. Jessop (22) at the discussion on Shell-Shock, without Visible Signs of Injury, at the Royal Society of Medicine. Jessop there stated he had had under his care numerous cases of temporary blindness

following the explosion of shells: "the blindness lasted under a week, and generally about two or three days. Both eyes were always affected, the pupils were active to light, and the ophthalmoscopic appearances normal. The fields of vision were sometimes contracted, but more often normal; in only two cases did I find a spiral field." It is to be regretted that more details were not recorded, especially as it is not clear exactly what explanation Jessop gave of their occurrence. He said that "the windage of the shell, or, in some cases, *the actual blows from falling down or violence* (italics mine), affected the cortex cerebri." If this were so in the two cases specified, then undoubtedly they had for basis an organic condition, presumably a cerebral concussion or commotio (the discussion dealt solely with cases without visible injury). In the absence of further information it is idle to speculate, but the assumption is perhaps justifiable that Jessop inclined to a view of their development which was not the ordinary theory of traumatic hysteria

The difficulty that surrounds the subject will be obvious to the reader. Under what circumstances certain side-to-side injuries of the back of the head produce constricted fields is not known; why my Case 1 shows the condition in typical guise while other occipital lesions that have come under my observation, by no means dissimilar in extent or site, are not followed by such clinical symptoms, is far from clear. For the present, it appears a reasonable contention that in certain cases without permanent visual defect of the accepted organic type of hemianopia or scotoma constriction of the fields, and helicoid or spiral fields, may be the expression of an organic change, the basis of which is a violent commotio, or a concussion amounting to contusion, of the visual cortex or some part or parts of it not more closely to be specified, or of the subcortical visual projection system.

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E. Treacher Collins.

Subchoroidal hæmorrhage from concussion and its relation to (a) *"commotio retinæ"*; (b) *"holes at the macula"*; (c) *pigmentation of the retina*; (d) *pigmentary disturbance at the macula.*

(With Plate I, fig. 1.)

Subchoroidal hæmorrhage is a condition with which ophthalmic surgeons are most familiar when it occurs after intra-ocular operations, such as extraction of cataract

or iridectomy for glaucoma. Presumably in these cases the choroidal vessels are sclerosed and brittle, so that they easily rupture, when, from diminution of intra-ocular pressure, the blood rushes in and expands them. The pressure of the effused blood in these cases is such as to force the choroid inwards, and the contents of the globe forwards towards the wound, from which some of it often escapes.

Subchoroidal hæmorrhage is also of frequent occurrence in cases of indirect rupture of the globe. In five specimens out of twenty of ruptured globe preserved in the Moorfields Hospital Museum, blood-clots are shown situated between the two outer coats of the eye.

That subchoroidal hæmorrhage may also occur in an intact eyeball, as the result of concussion, is demonstrated by a case which I recorded in the *Transactions* of this Society last year, the essential details of which are as follows:

Pte. T. H—, æt. 42 years, was, on February 14th, 1916, struck on his right eye by a piece of the case of a shrapnel. He thought it was a large piece that struck him, and that it fell down again. He immediately lost the sight of the eye, and it had remained blind up to the time I saw him, nine days later. There was a bruised area at the lower and outer part of the globe, but no perforating wound. The pupil was eccentric, being displaced upwards and inwards; it was only very slightly active to light. The anterior chamber was shallower than normal, and contained strands of blood-clot. By focal illumination a large blood-clot could be seen in the vitreous. By reflected light hardly any red reflex could be obtained. The tension was − 1. The eye was excised a fortnight after the injury. A description of its pathological appearances are recorded in *Transactions of the Ophthalmological Society*, vol. xxxvi. The feature of the specimen, of which I now wish to give a more detailed description, is the subchoroidal hæmorrhage:

After the eyeball had been hardened and frozen it was divided by an anterio-posterior section, passing obliquely

downwards and outwards through the seat of injury. Each half of the globe then showed a layer of blood-clot between the choroid and sclerotic. Below, it extended from about the centre of the ciliary muscle to within two discs' breadth of the optic disc. Above, from the centre of the ciliary muscle to a little behind the equator. The layer was thickest, both above and below, a little in front of the equator, where it measured 1·5 mm. in width. From that position it tapered off gradually in thickness anteriorly and posteriorly. Sections of the eye examined microscopically showed that, where the layer of blood between the two membranes was thickest, the choroid had become much thinned, and that this thinness was due to compression of its blood-vessels, their lumens had become obliterated, so that no blood could be seen in them. Where the layer of blood-clot thinned off, there the vessels of the choroid, filled with blood, began to appear (Plate I, fig. 1).

This specimen shows that blood effused from ruptured choroidal vessels beneath the choroid, in an intact eye-ball, tends to accumulate as a flattened layer, rather than in the form of a localised prominence; and that it compresses the choroid. The form which it takes is evidently due to its meeting with resistance from the sclerotic externally, and the tense elastic lamina and the intra-ocular contents internally.

Subchoroidal hæmorrhage must be differentiated from intra-choroidal hæmorrhage. The former is probably due to rupture of the larger vessels in the outer layer, and the latter to an effusion from the capillary or inner layer. The ophthalmoscopic appearances of intra-choroidal hæmorrhage are well known, and have been pictured in Leibreich's *Atlas*. It is the ophthalmoscopic appearances which may be produced by subchoroidal hæmorrhage that I now propose to discuss.

An effusion of blood situated external to the choroid would not be visible as such ophthalmoscopically, being hidden from view by the reflex from the choroid and

PLATE I.

FIG. 1 illustrates Mr. E. Treacher Collins's Contribution to the Discussion on Concussion Injuries of the Visual Apparatus (p. 112).

It shows a section of the choroid and sclerotic, with a subchoroidal haemorrhage the result of a concussion injury. Where the layer of extravasated blood is thickest the choroid is seen to be compressed, and its bloodvessels devoid of blood, elsewhere they are well filled.

FIG. 2 illustrates Mr. M. S. Mayou's paper on (?) Colloid Nodules in the Choroid in a Girl (p. 227).

Fig. 2.

Fig. 1.

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pigment epithelium. My specimen shows that an effusion sufficient to compress and empty the vessels of the choroid may occur, without giving rise to much elevation in the level of the fundus. A raising up of the choroid to the extent of 1·5 mm., as in my specimen, would not be likely to be detected ophthalmoscopically.

The ischæmia resulting from the compression of the choroid is the change which would be most likely to cause alterations in the appearance of the fundus. It, therefore, is necessary to consider what is the result of an acute ischæmia of the choroid. The well-known experiments of Wagenmann (1) on rabbits afford evidence on this point. In order to study the influence of the choroid upon the retinal nutrition he divided a single ciliary artery. Ophthalmoscopically, at first there was no change, but in the course of half an hour a grey white opacity showed itself in the periphery of the fundus, which rapidly spread through all that portion of the retina lying over the region of the choroid supplied by the divided artery. The opacity lay in the retina, not in the vitreous. It became whiter in course of time, attained its maximum in about six hours, and remained unaltered for a day or two. As it cleared off it assumed a reticular appearance, and small pigment spots became visible. These pigment spots increased in number and size, and the fundus assumed a yellowish hue, over which the retina could be seen as a delicate veil. The pigmentation increased up to the third or fourth week, when the appearances simulated those after choroido-retinitis.

Microscopically, the changes in the retina were seen to be most marked in the bacillary and the outer nuclear layers. They consisted in an effusion of an albuminous fluid, with molecular haziness and swelling of the tissue elements. The rods and cones were separated from each other, raised up from the limitans, and pushed about in all directions. Similar changes took place in the nuclear layers; an amorphous albuminous effusion lay between the bacillary layer and the pigment epithelium, filling up

the groove between the folds of the retina. Degeneration of the pigment epithelium occurred early, and changes like those in the outer layer (but less marked) were seen from the very first in all the inner retinal layers.

From these experiments we have the sequence of events which follow the permanent cutting off of the choroidal blood supply; they may be summarised as follows—a white haze of the retina, followed by pigmentation and choroidal atrophy.

The obstruction to the choroidal circulation, which is produced by subchoroidal hæmorrhage, is not necessarily permanent. The effusion of blood which causes it must cease as soon as it is produced. When the vessels become occluded no more blood escapes. The blood will then gradually become absorbed, and as it disappears the pressure on the choroidal vessels will be relieved. The upraising of the choroid compresses the intra-ocular contents, and escape of fluid from the vitreous would probably to some extent relieve the pressure on the inner surface of the choroid.

In considering, therefore, changes in the fundus likely to be produced by subchoroidal hæmorrhage, we have to differentiate the effects produced by temporary and permanent obstruction of the choroidal circulation.

As we have seen, the first change in the rabbit's eye is disturbance in the nutrition of the outer layers of the retina, resulting in œdema and opacity of that membrane. If the circulation soon became restored this œdematous opacity might clear up without any further changes ensuing.

Clinically we not uncommonly meet with cases of localised opacity of the retina following concussion injuries, which, after lasting a few days, completely clear up. The condition was named by Berlin (2) "Commotio retinæ." His description of the clinical appearances of an eye so affected is as follows: First symptom, considerable diminution in the acuity of vision in the centre of the field and marked resistance of the sphincter pupillæ to the influence of atropine. Within a short time a dis-

tinct haziness of the fundus over a considerable area becomes developed; sometimes small hæmorrhages result. The changes are most marked around the yellow spot; in one case there were two separate patches. The ophthalmoscopic appearances reached their highest development in twenty-four to thirty-six hours and disappeared in two to three days, in proportion to the violence of the injury inflicted. The sight improves greatly at first and then remains stationary, some defect remaining after the retinal haze has vanished. The deficient reaction of the pupil to atropine lasts about as long as the defect of sight.

Haab (3), in 167 cases of concussion of the eyeball, found 82·6 per cent. which showed no alteration on the fundus. In 4·7 per cent. of the cases there was Berlin's œdema of the retina in the macula region only, and in 12·5 per cent. it existed both in the macula and elsewhere. In 16 of the latter cases the changes disappeared without leaving any traces behind. In 5 cases there was persistent pigmentary disturbance at the macula, in 1 atrophy of the optic nerve, and in another some choroidal disturbance.

To determine the cause of the changes he had observed, Berlin experimented on rabbits, producing concussion by striking their eyes with an elastic stick. The change produced, to which he attached the most importance, was an extravasation of blood, which he always found, between the choroid and sclerotic in the situations of the retinal opacity. It always formed between the vascular and fibrous membrane a layer equal in extent to the opacity. Berlin concludes from this constant coincidence of hæmorrhage under the choroid with opacity of the retina, that the latter is the result of a serous infiltration of the retina of which the effused blood beneath the choroid is the cause. He also found an effusion of blood of less extent between the sclerotic and ciliary muscle and anterior part of the choroid.

These findings of Berlin's are so completely in accordance with what my own pathological observation would lead me to expect, that, notwithstanding Denig and Bach

having failed to find subchoroidal hæmorrhage as the result of their experiments, I think Berlin's conclusions for his own cases must be regarded as correct.

In the choroid the capillary plexus is finest in the macular region, and arrest in the circulation would, therefore, be more easily brought about in that situation than elsewhere, as a result of pressure from subchoroidal hæmorrhage. The retina at the macula is practically entirely dependent for its nutrition on the choroidal circulation. An interference with the circulation in that region would be more likely to affect the retina than where it has another source of supply.

As already pointed out, Berlin's œdema of the retina is more frequently found at the macula than elsewhere. Another change occasionally found at the macula following concussion injury is, what has been described as, "a hole of the macula." In describing the pathology of this affection, to which he devoted considerable attention, Coats (4) says: "Macula holes are produced by an œdema of the retina at the posterior pole. The œdema may not be confined to the region of the fovea, but the appearance of a hole will only be produced if there is a defect, at least, of the inner layers of the retina. Possibly, for the completely typical picture, without membranes or shreds, a total defect of all the layers of the retina is necessary, and that such a complete defect may arise from retinal œdema is proved (by his Case 2). The œdema may result from a contusion, in which case it is the same as the œdema which produces Berlin's opacity, or it may arise from toxines in the vitreous, the result of irido-cyclitis, or from retinal vascular disease."

It seems, then, highly probable that the conditions known as "commotio retinæ" and "holes at the macula," following concussion injuries of the eye, may both result from a temporary ischæmia of the choroid, caused through compression of that membrane by subchoroidal hæmorrhage.

The experiments of Wagenmann on rabbits show that ischæmia of the choroid of a lasting character is followed by pigmentation of the retina in the form of dots or a

network. Cases of pigmentation of the retina following concussion injuries of the eye have frequently been recorded. Brailey (5) published two cases in 1876, one in which the eye had been injured by a falling rocket-stick, and the other, of which he described the microscopical appearances, where the injury had resulted in a perforating wound followed by a traumatic cataract twenty-two years previous to the removal of the eye.

Jonathan Hutchinson, senior (6), recorded a case in 1888, and Jonathan Hutchinson, junior (7), described two cases and read a paper on the subject before this Society the following year.

Similar cases are recorded in our *Transactions* by Lawford (8) in 1902 and Bickerton (9) in 1904. I have met with several cases of the sort myself, one of which I am able to bring before you at this meeting, the details of which are as follows:

Albert A—, æt. 15 years, came to Moorfields Hospital on December 11th, 1916, stating that two days previously he had been struck in the right eye with a football, and that since then the sight of it had been defective. On testing his vision it was found that he could only see two letters of $\frac{6}{12}$ths with the right eye, whilst he had full vision with his left. Ophthalmoscopically there was noted, hæmorrhage into the vitreous and some hæmorrhages in the upper and outer part of the retina. A week later the blood in the vitreous had considerably cleared away, though there were still some large floating opacities present. At the upper and outer margin of the disc, adjoining one-fourth of its circumference, there was an area of the fundus appearing abnormally pale in colour: it was not bounded by any sharply-defined margin, but merged gradually into the normal fundus reflex. External to this pale area were a number of circular, pigmented dots in the retina, definitely localised to a sector of the fundus lying up and out from the disc. The hæmorrhages in the periphery of the retina up and out, which were noted when he was first seen, were still present.

On December 28th, nineteen days after the injury, the pigmented dots were observed to be darker in colour; some of them were seen to be definitely in front of the retinal vessels. The field of vision was taken and found to be of normal dimensions. The central vision had improved to $\frac{6}{9}$ partly.

A paper, in which are collected the notes of several cases of the same description, together with two new ones, by Marie Huguenin (10), appeared last year. Following Siegrist's (11) example, she described them under the title "Traumatic Rupture of the Ciliary Arteries." That these cases were due to rupture of a ciliary artery seems to have been inferred from the fact that the symptoms were those likely to result from a cutting-off of choroidal blood supply; it was not based upon pathological investigation. Indeed, Siegrist himself states that, "other causes than laceration of a ciliary artery can produce the ophthalmoscopic appearances he has described, as, for instance, a choroidal or subchoroidal hæmorrhage resulting from lesions of the choroidal vessels." "Such hæmorrhages," he says, "naturally influence the choroidal circulation and may produce secondary degenerations in the outer layers of the retina."

That concussion injuries of the eye may result in extra-ocular rupture of a ciliary artery yet remains to be proved, whereas there is definite pathological evidence that they may produce subchoroidal hæmorrhage. As ischæmia of the choroid, resulting from either lesion, may produce pigmentation of the retina, it is, I think, fair to assume that when pigmentation occurs in connection with concussion it is due to the lesion which has been shown may follow such an injury.

Following a severe contusion of the eye there is another change sometimes met with at the macula which may make its appearance after that of commotio retinæ has passed off; it consists in pigmentary disturbances of a varying degree, giving rise to permanent impairment of vision. Haab (12), who has directed particular attention to these

cases, in the course of five years met with 80 cases in 29,437 patients. He states that a more forcible contusion injury is required for its production than that which gives rise simply to Berlin's œdema of the retina. A description of these cases is summed up by him in his small atlas as follows: "After severe external violence from a blunt body, as a fall on the eye, a blow of the fist, a blow with a hammer, the wadding from a blank cartridge, an arrow, a blow of a whip, etc. The foveal region in the retina may show apparently slight alteration that may lead to severe and often incurable visual disturbances, and often assume great medico-legal significance in a suit for damages. The mottling and pigmentation are often very insignificant, especially in the beginning, and may continue so throughout the disease, or may be replaced by more distinct pale patches. Sometimes there is only slight separation of the pigment confined to a small portion of the middle of the foveal region, such as occurs in myopic or senile macular disease; unlike Berlin's traumatic opacity of the macular region which disappears, the pigmented patches of this disease are permanent."

So far the pathology of these pigmentary macular changes, the result of concussion, has not been worked out. I suggest, as a plausible hypothesis, that they also, like pigmentary changes in the fundus elsewhere from concussion, may result from ischæmia due to subchoroidal hæmorrhage.

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Capt. CRUISE

said he thought members of the Society were much indebted for the way in which Capt. Ormond had described the manifold signs and symptoms which his experience at the 2nd London General Hospital had enabled him to study in regard to injuries inflicted in warfare. It opened up considerable knowledge for those who had had nothing approaching that gentleman's experience.

In the 3rd London General Hospital, where he was himself working, there were 200 beds, and he had seen a fair amount of these injuries received in warfare. It was difficult to keep accurate notes of the cases, because his assistants had been varied and migratory, and therefore, he had to speak on his general impressions.

Capt. Ormond had collected a most valuable and interesting series, and if he, the speaker, might criticise him in a friendly way, he would refer to the question of "windage," on which Capt. Ormond asked for expressions of opinion. On looking through the table of cases which were supplied in connection with the paper, he felt that the evidence on which the effect of windage was presupposed was not at all convincing. In studying these cases on this side, the evidence tendered, especially by the patients themselves, must be examined and sifted with great circumspection. The cases when seen over here had had the injury some time, the interval varying a good deal, and the evidence given by the man himself he regarded as quite worthless. At the time of the

mishap, the man had not the slightest idea of what happened to him, and by the time he arrived in this country he had probably concocted a plausible and continued narrative. A case which showed a striking example of that was that of a man who came over in the first three months. He was lame, but could not understand why. The hospital had a most enthusiastic radiographer, and a skiagram was taken of the man's foot. There was no history of any action going on, and there had been no chance of his being hit according to his own story. The skiagram revealed a rifle bullet lying underneath the metatarsal joint. There was no external trace of injury, and the man would not believe it was his foot which had the bullet in ; he thought the plates had got mixed up. He denied that he could have been hit, or that there was any firing taking place where he was ; he only knew that he went lame, and he thought it was due to a corn. In Case 12 of Capt. Ormond's series, the evidence again was doubtful. He admitted cases of injury due to windage did occur, but not with the frequency which was suggested by giving eleven cases out of a total of eighty. Case 12 did not lose consciousness, but he had two small scars, one in the right upper lid, and one in the lower lid. Those seem to afford a much more likely explanation of the injury than the rather problematical one of air concussion or "windage." In Case 15, the man was seen more than twelve months after the injury. The man's own history had to be relied on, and it seemed much more probable that he had a blow on his eye from a clod of earth, or some other material, which was answerable for his condition. Case 57 was impossible to him, and apparently Capt. Ormond himself was doubtful about it. He did not think the note given could be regarded as circumstantial evidence of windage effect. Since the question of concussion injuries came up, he had been looking out for these cases, but he had not seen cases attributable to that which had convinced him. It was most important to have the question discussed.

With regard to injuries to the retina, he had been much struck by a series of cases he had had, in both officers and men, of "through and through" injury, and his conclusion was a contradiction to Col. Lister's view that there was practically no damage resulting to the eye unless the body passed through the orbit. He had had several cases of through and through injury below the malar region, or injuries which did not involve the orbit, the bullet having struck one eye, and passed tangentially through and come out in the region of the nose or inner canthus, and in which there had been this type of injury in the other eye. He noted the case of a lieutenant, who was struck six months ago, the bullet passing through the temporal region, above the zygoma, and coming out at the right eye. That man had got the same type of injury. The lesion consisted, essentially, of fine hæmorrhages, small and punctate, at the macula, varying from an appearance which one would describe as barely more than physiological, to an organising mass in the region of the macula. Those were indirect concussion injuries. There were minor ones, in which nothing could be made out except a central scotoma, a serious result of showing a minimal objective lesion. The bullet had passed through the cheek, or some neighbouring cavity, and there was no other injury to the orbit or to the movements of the eye, though one would have expected a difficulty in depressing the eye. He had watched the hæmorrhages, and the improvement had been very small: they went on to pigmentation, and, possibly, cystic degeneration at the macula. This was a true direct effect. And the explanation of them was, probably, to be sought in the peculiarity of the blood supply in that region. The explanations given were not very satisfactory. They were not *contre coup* effects, in which one expected the effect to take place chiefly at the opposite pole to the injury.

Capt. Ormond also referred to hæmorrhage into the cornea. He, the speaker, learned about the physiology of the blood, and about hæmatoidins and hæmosiderin being

deposited in the lamelæ of the cornea from Mr. Collins. In most of these cases there was a greatly increased tension in the eyes. He had had two cases in which that tension was not present, and the eye was quite pulpy. The anterior chamber had been full of blood, and the cornea had absorbed it. He had one man now in the Hospital, whom he showed to Capt. Parsons, who, on looking at it superficially, in a somewhat indifferent light, thought it was a congenital cataract. In three of his cases the eye had been very soft and pulpy, so much so that he took one eye out, on account of the risk. The other two he had been watching for some time. They did not always clear from the periphery; the last one he referred to cleared in a sector-shape from the side.

Detachment of the retina was also referred to in the paper. He had been very much struck with the absence of any considerable number of cases of detachment of retina. He thought the majority of members of the Society would agree that in the collection of eighty cases which Capt. Ormond had brought together one would naturally look for detachment of retina in a larger number than eight, or 10 per cent., certainly a larger proportion would be expected to occur in civilian cases. Only three of the eight were detachment in the ordinary sense he was now meaning; he did not refer to the subretinal or subchoroidal hæmorrhage causing separation of the retina, but a detachment due to a blow, a floating detachment, with a tear in it. A discussion on detachment of the retina was held some time ago, and he thought it important that these figures should be remembered, as not contributing to the general ophthalmological impression that a blow on the eye was very liable to produce detachment of the retina. He did not know whether it was due to the high velocity of the projectile causing a tear, and subsequent adhesion between the layers, and whether slighter blows had a greater tendency to cause detachment. It was not yet known how many more would be seen on account of shrinking

bands, but the actual occurrence of detachment of the retina seemed to be comparatively infrequent in the war.

Sir GEORGE BERRY

said, in response to the President's invitation, that he had come to learn. He had seen many of these cases, but he had not formed a very definite opinion about them, nor had he made any proper collection of them.

There were certain points which had interested him very much, more particularly the one the President had just told the meeting about, viz. the condition known as commotio retinæ, and the pigmentation which took place afterwards, in many cases without any definite ophthalmoscopic appearances being noticeable some time after the accident. He would like to hear from the President whether his explanation also accounted for the very great degree of pigmentation which might occur after a subchoroidal hæmorrhage, he meant cases in which one saw these acute so-called concussion wounds, followed, a year afterwards, perhaps, by coal-black masses of pigment.

Another matter which interested him in Capt. Ormond's contribution was the statement that in all cases in which there were permanent hemianopic symptoms there had been a degree of blindness to begin with. That had been entirely his own experience in a considerable number of cases of hemianopia of all kinds which he had seen as the result of bullet wounds in the occipital region.

Mr. A. L. WHITEHEAD

said he saw, a short time ago, a very interesting case of commotio retinæ. He saw the case within an hour of the accident, and there was complete commotio retinæ—absolute pallor of retina on one side, slight pallor on the other. Vision was reduced to ability to see the hand move when held quite close to the face. After a very short time, however, the vision began to improve, and in a fortnight it had been quite recovered. He was unable to conceive

that that could have been caused by subchoroidal hæmorrhage, a hæmorrhage which produced practically complete commotio retinæ, which disappeared in a fortnight, leaving no trace, and practically no alteration of vision. He thought it much more likely to have been due to molecular changes in the retinæ, though on that point he was open to correction.

With regard to rupture of the retina, he had seen one military case in which there was an extraordinarily beautiful picture of ruptured retina. There was no other injury to the fundus, except exactly in the macular region, where there was a minute oval puncture. This caused serious interference with the vision, but there were no other changes. He agreed with Capt. Cruise that uncomplicated detachment of retina in military cases was very rare. He only remembered two or three uncomplicated cases, of ordinary civilian type, following traumatism.

With regard to the pigment changes, there was a particular pigmentation which he had noticed frequently, and which he was in the habit of calling "pigment migration," *i. e.* an appearance suggesting that pigment had moved away from certain more or less circumscribed areas, and had piled itself, or had been irregularly distributed, at the side of the more atrophied area, without definite hæmorrhages This did not at all resemble the large masses or lumps of pigment which were seen following hæmorrhage. He had noticed it in cases in which there had been but slight superficial injury to the eye, and cases which Capt. Ormond described as "windage" cases. Nearly all these patients told one they were knocked down, or fell down, when the shell exploded near them, though there had been very little direct injury to the eye. He had noted this pigment migration in those cases.

He had also noticed, in cases in which there had been very slight injury of the eye, a variable state of the accommodation; in some there was an irregular spasm of accommodation, leading to apparent myopia, which passed off

on the use of atropine. In other cases there had been a variable irregular paralysis or paresis of accommodation, which ultimately cleared up. Probably those cases were largely functional, but he thought them worth mentioning as complications which might arise in concussion. (The PRESIDENT: Were the cases associated with minus tension?) So far as he could recall the cases were not associated with much lowering of tension, still less of collapse of the eyeball. (Mr. WHITEHEAD: It was just such cases that I referred to when I spoke of spasm of accommodation.)

Dr. GEORGE MACKAY

said that a question upon which he would have liked to have heard something in the discussion, but had not yet done so, was that of traumatic myopia. Many members must have seen, in civil life, *e. g.* in connection with gunshot injuries, cases in which the patient's refraction was profoundly altered and rendered, temporarily, myopic, perhaps to — 7 D. or — 8 D. sph., by a blow, especially on the anterior half of the globe. He had usually regarded this as due to a turgid condition of the ciliary body, causing an enforced and sustained accommodative act. He would like to know whether there had been many cases of that kind seen by the surgeons present who had been closely connected with military work in this country. He thought one reason why they had possibly not been seen was that they were, for the most part, transient, so that those surgeons who worked nearer the firing-line had a better chance of seeing them than those in the British Isles. He asked whether the President or any other member could supply information as to the pathology of this condition; whether there was simply a local œdema, or how the myopia was produced.

Mr. HARRISON BUTLER

said he knew very little about the subject, but at Birmingham Mr. Jameson Evans had been trying to collect records of cases of concussion injuries without a

blow on the eye. He believed that he had succeeded in collecting two cases.

In reference to the point raised by Dr. Mackay, he had seen several cases of blows on the eye followed by a myopia which lasted two or three days. He had thought it was due to an alteration in the position of the lens. It had been very transient.

Mr. Cridland

said that since the title of the discussion was announced, he had been looking for evidence in the fundus of the effects of very slight blows on the eye, and he had noted how frequently fine striation at the macular region was to be seen if carefully looked for.

The condition appeared within twenty-four to forty-eight hours after the blow, lasted for from seven to ten days, and then cleared up leaving no traces.

The vision was not involved until the striation appeared, but it was then in most cases reduced to $\frac{6}{9}$ or $\frac{6}{12}$, improving as the striation waned and finally returning to normal.

He did not consider that the appearance fitted in with the description of that known as Berlin's opacity in that it was not so marked, but he asked whether it should be regarded as a mild form of that condition or of the condition known as commotio retinæ, which he always felt was rather vague.

The point he wished to emphasise was the frequency with which striation at the macula of the "rising-sun" type occurred in his experience after very slight blows on the eye.

Mr. N. Ridley (Leicester)

said he had not looked up these cases, but he had noticed there were many cases of injury to one eye, resulting in total loss, and smashing of the face by bullet or shrapnel, where the other eye had escaped injury altogether, yet the concussion must have been tremendous.

Capt. ORMOND

said he would be very glad if the President would tell the meeting something of his knowledge of concussion cataracts, as he believed Mr. Collins had cut sections of such. Could he say where the change took place, and what was its probable explanation? The cases were extremely interesting ones, and differed from ordinary cataract.

The PRESIDENT

said he had cut sections of some eyes which had concussion cataracts, and in these he had found that there was rupture of the capsule. It did not, of course, follow that this was always the case. The cases of Vossius's concussion cataract could not be due to rupture of the capsule, nor those in which there was only a star-shaped figure at the posterior pole. He believed these star-shaped opacities were due to a separating out of the segments of the lens, and that the star-shaped figure was that of the segments of the lens with albuminous fluid collected between them, causing irregular refraction. He did not think there had been a concussion cataract examined microscopically in which the capsule was not found ruptured. In cases of concussion cataract of slight degree the opportunity of microscopical examination seldom occurred. When rupture of the capsule occurred it was generally at the equator of the lens. He had examined one case in which the rupture of the capsule occurred at the posterior pole, and the remarkable fact about that case was that for several days there was scarcely any opacity of the lens, although the lens matter was mixed with the vitreous.

Capt. ORMOND

(in reply) said he was glad to hear Capt. Cruise's remarks, as he wanted the opinion of members on the question of "windage"; it was largely for that reason he had brought the subject forward. It was impossible to be sure whether force exerted through the air could produce any real discontinuity or rupture of the tissues.

An excellent instance of the unreliability of a patient's story was given in Mr. Lawson's paper on "Detachment of the Retina at the Ora Serrata"; there was very great liability for the patient to be mistaken. If there were tiny scars on the cornea they were easily seen, and careful search sometimes gave a clue. Case 12 was looked upon as being one due to "windage." There was nothing apparent on casual inspection, but when he, the speaker, examined the skin minutely to see whether any scarring had taken place, he was satisfied there was evidence that the cutis had been ruptured. It must still remain an open question, but he hoped those present would see if they could obtain more information on the subject. General literature certainly assumed that these things existed, that "windage" caused definite lesions, an assumption which he thought had not been proved. The most satisfactory case was that of the man who, although at a very considerable distance from the bombardment, did have a definite rupture of a vessel, and a vitreous hæmorrhage, and it was impossible, as far as one could tell, that he could have been directly hit. He did not want members to conclude that he was convinced about the matter, or to think he did not realise that the evidence was extremely faulty, and should never be accepted on the unsupported statement of the patient himself. With all the best intention in the world, these men were sometimes mistaken.

With regard to detachment of the retina, Mr. Nettleship worked at the subject a long time ago, believing that in these cases detachment of the retina was not very common. Pathologically, the retina was so matted to the choroid that detachment was not likely to take place as a result of the severe contusion injuries. Of his own cases, four were what he called solid detachments, due simply to large hæmorrhages underneath pushing the retina forward, and two of the remaining four were those in which the detachment came on some months later, in myopic eyes. He had had two cases of that, and two in which the eye was not myopic.

He thought the meeting would feel much indebted to the President for his explanation of commotio retinæ; it was a very valuable contribution and a suggestive one as to what possibly happened in these cases. The only difficulty he felt was that which Sir George Berry suggested, namely, that it was a little difficult to suppose that a subchoroidal hæmorrhage did produce those very extensive cases of pigmentation in which almost the whole of the inner and upper, or the upper and outer part of the retina, was involved in a definite pigmentary change.

With regard to rupture of the ciliary vessels, in several of the cases he had recorded the cause was a bullet having passed through the apex of the orbit, not severing the nerve, but passing close to it, and, probably, close to the posterior pole of the eye. He thought some of the ciliary vessels might have been ruptured.

He had not seen any cases of hole in the macula which represented rupture of the retina alone. But he thought that he might see some later, because in one or two cases in which he expected that he would see it he had not done so, probably because the time had not yet elapsed. From what Mr. Coats had written and the President said, these holes might appear after a lapse of time; at the time the cases were seen there might be merely pigmentary changes and local disturbance.

With regard to traumatic myopia, which Dr. Mackay mentioned, he feared that the speed with which the work had to be done at the military centres did not allow of the examination of cases, likely to be transient, with the care that Dr. Mackay's leisure permitted; but, no doubt, there were a large number of cases in which the refraction was altered appreciably. It was necessary, in the stress of military work, to take the attitude that unless a man had got a very definite lesion, the less the attention of the patient was drawn, by examination, to the effect that he had something wrong with him, the better, though the scientific attitude might be a different one.

IV. DISCUSSION ON THE EMPLOYMENT OF THE BLIND.

Opening Paper.

By Sir A. Pearson.

The first part of the afternoon was given up to a visit to St. Dunstan's College for the Blind, Regent's Park, which is under the control of Sir Arthur Pearson, Bart., himself sightless.

After an inspection of the varied crafts and other accomplishments being taught in the College, including the keeping of poultry,—

Sir Arthur Pearson said: I am very glad indeed to have the honour of opening this discussion, and to tell you something about the fellows who have lost their sight in the war and are making good, and getting even with their handicap in so very remarkable a manner. St. Dunstan's has set an entirely new pace in the blind world. As an illustration of that, I do not think I can do better than tell you of an occurrence which happened here six or eight months ago. I had a letter from the Chairman of Henshaw's Blind Asylum, which is the biggest institution for blind people in England, and one of the best. The Chairman asked me in that letter if he could bring a deputation to London to consult with me on an important matter. To this I gladly agreed, and when they came the spokesman said: "We have been looking at the fellows whom you have trained at St. Dunstan's and have settled in our part of Lancashire, and we have come up from Manchester to ask you to tell us how it is you can teach a man, in seven to eight

months, as much as it takes us four or five years to teach him." It was a curiously interesting question, and it went further than the spokesman said, because, besides teaching the blinded soldiers the industries which had so impressed him, they had also learned the difficult task of reading Braille very well, to use the typewriter with speed and accuracy, netting, and other things which it is good for a blind man to know. I replied: "There are several reasons, and I will tell them to you. First of all, abolish from Henshaw's that dreadful word affliction, and with it the morbid and depressing thoughts to which it gives rise. By doing this I am sure you will reduce your training time by one half. If you tell a man he is afflicted, he becomes afflicted, mentally and physically; but tell him he is handicapped and show him how to get over it; he becomes interested, his spirits rise, and he tastes the joy of achievement." He finds that he can do things, and how much easier it is to do them than he thought it would be.

Another potent factor here is the employment of the blind teacher. I think you will realise how intensely encouraging it must be to the blind man to find he is being taught by one who is himself blind. A sight teacher may say to the pupil, "Here is the way to do it." The blind man will say to himself—he may even say it to the person who is talking to him—"That's all very well, but what do you know about it?" And he is right. But when he is being shown by a blind man, he feels that he is being taught by the best methods, and says to himself, "What this blind man can do, I can do." We develop that feeling by the employment of the blind pupil-teacher; we select the most apt men for these posts, and pay them regular wages. And the first thing that happens to a fellow when he comes here helpless and hopless is, that he is taken hold of and shown to do something by one who was himself blinded only seven or eight months ago. You cannot think of anything more encouraging and more apt to lead to good results.

The third point, which is of very great importance, and which seems contrary to all that one would believe to be right, is the very short hours during which the men here work; they work only two and a half hours in the morning, and two hours in the afternoon. Those who are in the class-rooms in the morning, learning Braille and typewriting and netting—which is not taught as an industry but as a paying hobby—are in the workshops in the afternoon, or down at the poultry farm. These newly-blinded men become brain-weary if they stick too long at their work. The shortness of the hours of study is one of the main factors which go to secure the celerity with which these men learn to re-equip themselves for the battle of life; in fact, learn to be blind. There is something which is difficult for a seeing person to understand about the difficulty which these experience in learning. It is the intense mental application required when the sight has been lost; one has to be always thinking. I can move about this place perfectly, and in my own home too, anywhere, in fact, in familiar surroundings. But if I am walking and at the same time talking to somebody, or even if I am thinking about something except the way I am taking, I shall very likely make a mistake. Probably one gets the better of this as time goes on, and that intenseness of application becomes less and less necessary. But the recently blinded man has to keep his mind tuned up to a high pitch, and this soon produces a feeling of mental weariness. During the seven or eight months a fellow is here, he learns to read Braille well, and at a speed sufficient to enable him to enjoy a book. I am insistent upon a man doing everything he can to put himself back into normal life instead of being dependent upon others for almost everything.

Typewriting is not only useful, it is pleasurable. To instance my own case, I have probably written fewer letters than most men, because I have always dictated, except, of course, letters of a quite personal nature. But now I find myself getting up earlier in the morning

in order to enjoy typing a few letters. Besides having learnt to read and typewrite these fellows leave here, some able to manage a poultry farm, some to make things as carpenters, some to do boot-repairing, mat-making, or basket-making. Some have learned to write shorthand by the Braille system. All this they have learned in the almost incredibly short time of seven or eight months.

Masseurs take longer than that; the average is nearly twelve months; the time taken depends somewhat on the date of the examination which they have to pass, as the Incorporated Society holds these only twice a year. Massage is a part of the training here in which I think you gentlemen will be particularly interested. It has been a great success at St. Dunstan's, as has everthing else which we have attempted to teach the blinded soldier. Massage has, indeed, been an amazing success. At the moment forty fellows are learning it here, and they are trained very thoroughly. I think we have the best equipped school for massage in the kingdom. Our students have to pass the examination of the Incorporated Society of Trained Masseurs, which is the stiffest in the country. So far, everyone of our men who has been up for this examination has passed, and every man we have placed at a hospital has given such satisfaction that, with no exception, have come requests for more. I heard, only this week, from the officer commanding a hospital in the North, to say that the blinded soldier masseur whom we sent him is the best of their staff, and asking for three more. The Medical Officer who supervises massage at a large command depôt in Lancashire, where are 6000 men under treatment, told me that of the thirty-two masseurs there, the four blinded soldiers from St. Dunstan's were incomparably the best. This success has been, to me, amazing. These men, whose standard of education is by no means high, had to learn more than the rudiments of anatomy, physiology, and pathology. In the early days this branch of the work seemed to present difficulties

which appeared almost insuperable. The adoption of special methods and the determination of the pupils has led to the accomplishment of wonders. Obviously only certain men are fitted for this profession, and we are careful to pick out the right ones.

For massage one must have a fellow who is not disfigured, who is strong, healthy, and active, and who is well-mannered and intelligent enough to tackle the stiff task which is set him. With regard to shorthand, as written by the blind, experts will tell you that it takes four years at least to master the art. Our fellows here do it in seven to eight months. I have letters from employers where they have been placed, saying they are giving complete satisfaction, and this without making any allowance for their blindness: they are doing the ordinary work of shorthand correspondence clerks as well as it needs to be done. Everyone of them is earning as high wages as before he was hit, some of them higher. The masseurs are earning at least £2 10*s*. a week. In fact, there are a large number of blinded soldiers who have been trained at St. Dunstan's and who, besides being equipped with a useful and interesting occupation, are making more money now than they made when they could see. That is a wonderful thing to have happened. I should detain you too long if I were to go through all the occupations we teach here—carpentry, basket-making, cobbling—the workers at which are all earning good wages. I had a letter last week from a lad who had settled in Guildford as a cobbler; he said that for the first three months his average weekly earnings had been £1 9*s*. 1*d*. I could give you dozens of instances of the same kind.

A point which will interest you, gentlemen, rather particularly, is again a curious one in one respect but not in another. The fellows who get on best at St. Dunstan's, and have, from the start, are those who are quite young and quite blind. Those who possess a little sight do not do as well as those who can see nothing at all. There is

no one here who possesses any useful degree of sight. Our definition for admission here is, that a man should not be able to read and not have sufficient sight to enable him to pursue an ordinary avocation in the ordinary way. When I come across a fellow who can see a little I ask him whether, at arm's length, he can see if his fingers are open or shut. If he cannot, and is unable to read, he is technically blind. The majority of the fellows here have no sight at all: a third of them have no eyes, another third are dark blind, and of the remaining third, one-fifth or one-sixth have something more than mere perception of light. Those who possess a very little sight get on worst, and that has been so all the way through. I know, from my own experience, how hampering and unuseful it is to see a very little. Your business, gentlemen, is to preserve in any degree the sight of those who come to you. To some, and especially to old people, a modicum of sight is very precious. But you may take it from me it is right to tell the young or healthy people who are likely to do anything for themselves, or to carry on in the world, not to feel miserable because the little sight they have is leaving them; they will do very much better without it. People who can see use their eyes practically to the exclusion of the other senses; they almost leave their other senses to use themselves. It is only when the sight goes that the brain turns to the other senses and starts developing them. As you may know, there are some extraordinary fallacies about the quickening of the other senses when sight is lost. Nothing of the sort happens. When a person loses his hearing you do not expect him to become longer-sighted than he was before. So it is with the sense of smell, which is very important, the sense of touch, and the curious senses which develop when the sight is lost—senses of which you scarcely knew the existence. The principal of these are the sense of obstacle and the sense of direction. They develop in different degrees with different people: to some they come quickly, to others slowly, to others not at all.

It is important to teach the blind man not only to work, but also to play, so that he can re-enter normal life in every possible way. That includes play as well as work; it includes reading and typewriting, and playing games, and playing on some musical instrument or other. Most of our men are learning to play some musical instrument, from a tin whistle to a violin. Some are taking singing lessons.

I could talk to you for a long time on how fellows here work and what they do, and how they progress with their work and with their play, and in the word "play" I include exercise. If you were here after five o'clock you would see Regent's Park lake swarming with rowers from St. Dunstan's. But for blind people adequate exercise presents difficulties, because there are few forms of it which one can continue to pursue when sight has gone. There are five mainstays: walking, rowing, which the blind man can do as well as anyone else, whether he has done it before or not, swimming, to which exactly the same remark applies, tandem-cycling. They, however, give you a very fair range in the way of exercise.

Mr. W. C. Rockliffe:

In the first place I must thank the Council of the Ophthalmoscopical Society for the honour in their having selected me as one of those worthy to speak on this subject of "The Employment of the Blind," especially as the welfare of the blind has been one of the deepest interest to me for many years, and chiefly because these remarks will, I trust, be of some service in assisting to brighten the lives of our plucky and undaunted heroes.

As this short paper has been written prior to my knowledge of what Sir Arthur Pearson's opening paper on this discussion would comprise, I fear it will contain very few, if any, further additional suggestions.

On receipt of the communication from the Council of the Ophthalmological Society requesting me to open this

discussion on their behalf (as the matter seemed to me to be extraneous to the objects of our Society), I wrote the Secretary for an explanation, to which he replied that he wished me to treat the subject from a surgical point of view. I fear, however, from a professional point of view, there is not much to be said except that *mat* and *ship-fender* making, on account of the dust, is not an altogether desirable occupation, and I fear is somewhat deleterious to the health of the employees, as coir workers in consequence are frequently troubled with bronchial affections, presumably from the irritation of the fibrous particles, and, in addition, from the exertion necessitated in knotting the rope, they are always more or less emaciated as compared with the other trades, such as basket making, etc.

Bristle-brush making also causes, in some, irritation to the eyes, but to no great extent, from the fumes arising from the hot pitch. Willow-basket making, with spinal or abdominal defects, is also a source of trouble and rarely endured, especially in the larger rough class, such as coal baskets, etc., in consequence of their inability to bend when making the bottoms. Cane baskets of the lightest variety, such as fish-landers, etc., are more easily tolerated, being less arduous, but even this, in case of a man blinded from spinal injury, is trying when bending down to make the bottom of the basket.

With these few remarks I would leave this surgical aspect of the employment of the blind, except to add that, when selecting an employment for our blinded soldiers and sailors, their previous occupation, prior to joining the Service, should be taken into consideration; for example, one with previous outdoor occupation should not be trained as a shorthand typist or masseur, and not infrequently *vice versâ*, in consideration of their health.

Approaching this subject apart from the surgical point of view, in order that, if possible, I might further assist Sir Arthur Pearson's splendid and energetic efforts for these sadly afflicted brothers, I have been in communica-

tion with the secretaries and managers of fifty of the principal blind societies of the United Kingdom, having submitted my suggested list of employments, and requesting any further propositions, but regret that only six were able to make additional suggestions.

The following sources of employment were submitted to the various secretaries: Basket making (including willow, cane, and rush work); mat making; brush making (including broom, hair, and pile makers); boot and shoe making; rope, wire, and cord makers; cork-fender making; weaving; cane chair seating; knitting by automatic machine; net braiding; window-cord plaiting.

Special occupations for wounded soldiers and sailors.—Boot repairing; carpentry; poultry farming; dairymen and vegetable gardening; diving, for salvage purposes; telephone operator to private switch; shop keeping; hawking, including tea agencies; piano-tuning for the musical; blind visiting, libraries, collector of subscriptions, rates, and accounts; shorthand typist.

Further suggestions by: Mr. Thurman (Birmingham), insurance agent; Mr. Cleek (Leicester), teaching; Mr. Stone (Edinboro'), teaching; Miss Robinson (Wakefield) and Mr. Clements (Norwich), journalistic; Dr. Bainbrigge (Kent), permanent concert party dictaphone; and for the partially blind (with whom I have nothing to do), by Mr. Stone (Edinboro'), wireless operator and drill instructor to children.

Comments were made on my proposed occupations by the following experienced instructors of the blind:

Mr. Thurston (Birmingham).—I hesitate to suggest mat-making, because of the *trimming* of the mats. This, however, might be done if a blind soldier, practising this industry, happened to live in the neighbourhood of workshop for the blind where mat-making is practised.

Mr. Priestley (Bradford).—Lays great stress on the necessity for sighted supervision, adding it is his experience that home industries largely fail because there is no guiding hand in little things that turn up from time to time.

Dr. Rosendale (London Association for the Blind).—Ridicules the idea of any blind man being trained to earn his own living in three or four months to be of a permanent utility, and at once strikes off *all* my suggestions except piano-tuning, telephone operator, and blind visitor and collector.

Mr. Hardy (York).—There is an objection to dairymen and milkman from the point of view of the public health.

Mr. Stone (Edinburgh).—I should say boot-repairing, carpentry, poultry-farming, dairy-farming, vegetable gardening are all suitable and possible occupations, but I have found that *boot-repairing* is not at all a suitable occupation for a blind man in Scotland, although it may be in England. *Carpentry* as a *means of livelihood* not possible unless carried on in a large workshop. Poultry-farming, dairy-farming, and vegetable gardening require capital. Telephone operating, shop keeping, piano tuning, massage, typewriting provide a competent living. Piano-tuning is one of the best, but requires aptitude.

I fear and anticipate that Sir Arthur's address will have probably included most, if not all, of the above list of possible occupations, which of necessity must be lengthy, from which to select an appropriate employment in order that not only as a permanent, but lucrative occupation, of a lasting nature for body and mind may follow the training.

For, apart from blindness, there are difficulties to contend with—

(1) *Bodily deficiencies*, such as loss of fingers, limbs, and sense of touch.

(2) *The adaptability* of personal temperament, previous education, and social position.

(3) *The receipt of pensions*, which would naturally cause a dislike for laborious employment or the control and regulations of an institution.

(4) *The necessity for available assistance*, to ensure success in certain employments. Much stress of this

latter point is made by several of those replying to my letter, and which I fully endorse. Nevertheless, assuming a fair amount of competency and business capacity, telephone operator at a private switch ; piano-tuning, blind visiting, librarian, collector, etc.; massage, typist, and dictaphone, teaching, can certainly be carried out with the aid of a *guide only*, or even in some cases alone. If the blind man is blessed with a good wife, poultry farming, dairyman, and pig keeping, etc., vegetable gardening, I think the best. I happen to know one such man (blind), son of a farmer, who made sufficient as a dairyman, etc., to enable him to retire on a small income before he reached the age of fifty.

Re telephone work.—It seems to be impossible at any public up-to-date office, as the calls, etc., are made by coloured electric lamps, and, in fact, the sighted operators are required to pass a colour vision examination prior to election, and, as there are so few private switches, especially on the old system, this occupation must be limited.

Re massage.—Assuming the blind man is of good social position and education, and especially if he has previously been in gentlemen's service as valet, butler, etc., this is one of the best occupations, so much depending on the manner and courtesy when treating patients.

Re typist.—To a former such clerk, St. Dunstan's training is sufficient to ensure complete success, as I know from one case in my own city, where such a blind soldier has been reinstated to his old position at his old salary, and is giving satisfaction.

Re tuning.—If decided musical talent is present, the obtaining of a degree should, if possible, be taken.

But the greatest and most useful of all occupations (for pensioners) is undoubtedly, as Sir Arthur Pearson emphasised at the 1914 conference, the complete mastery of the Braille type, which, like swimming, skating, riding, etc., is never forgotten.

May I be permitted to say one word *re* "after-care,"

the latest of St. Dunstan's grand movements. I am not aware of the views, but it seems to me that if every man were allocated to the local institution in the situation of his own home, failing which, the division of the union of institutions, societies, and agencies for the blind in which he resides, all the necessary assistance could be obtained and would be gladly given, and he would feel he had someone near at hand to whom he could always refer for any assistance required; at the same time it would relieve the responsibility of the National Institution.

As representing the Hull Blind Institution, so long as I am officially connected therewith, it will be my own pleasure and privilege to do all I can to assist these patriotic heroes.

Mr. A. Lawson

said: As my contribution to this afternoon's discussion I want to emphasise the importance of certain features appertaining to the great question of the training the blind; and around the kernel of this discussion, viz. the best means of employing the blind so as to enable them to regain their independence and continue to be useful citizens, circle no less important points as to the methods by which these means are to be obtained.

Before the war these adjuncts to the actual teaching of handicrafts, etc., were, to a very large extent, neglected, not because they were not to some extent realised, but because the machinery to cope with them was not in being. We were at that time presented with the picture of a number of isolated societies, trusts, and workshops mostly doing excellent work, but acting more or less independently of each other. A certain number of blind charities existed, as they do now, dealing with one particular phase of the question, such as money grants; others specially directed their attention to literature for the blind; others furnished workshops for teaching handicrafts, and but very few attempted a systematic training of the blind in all its

varied aspects. In this way there was much overlapping and a great deal of inevitable inefficiency. The workshops were too few and badly placed, the means of obtaining the necessary funds to admit the poor blind for training and maintaining them during training were insufficient, whilst the training methods themselves were full of imperfections, largely the result again of money difficulties and ignorance on the part of the charitable committees responsible for the management.

The crowd of young blinded men now thrown on to their country has raised the question of the training of the blind to one of national importance, and brought into prominence the inequalities and inefficiencies of what one may now term "the old methods." The brilliant initiative of Sir Arthur Pearson, of which St. Dunstan's is the exponent, has, as the foundation of its success, the first great principle underlying the whole question—that of the centralisation of effort. That is, St. Dunstan's was intended to become, and has become, a centre where all aspects of training—moral, social, and economic—are fostered and encouraged. The absolute importance of this point cannot be over-estimated. We who have been working here since the start realise most forcibly what a large number of isolated questions are involved in this training of the blind. It has to be conceived in the first place that those who set out to train the blind must be themselves trained. Experience, in other words, is an essential factor, and this can only be obtained by time and by the practice acquired at a centre of training. The success so far achieved by St. Dunstan's is largely owing to the realisation by its founder that the blind man cannot be trained by giving him a pension and casting him loose to attend a reading-class here and workshop there, when and where he is so disposed or otherwise. It consists in forming a college where the blind man is received to learn first how to live as a blind man, to be encouraged and supported in his initial difficulties by the companionship and experiences of others similarly placed.

He is shown by trained nurses or nurse companions how to overcome the primary difficulties of dress, feeding, and the maintenance of his social independence, the loss of which has been hitherto one of the great stumbling-blocks in dealing with the blind. In the learning of trades and handicrafts he is taught to a large extent by those who are themselves blind, and who, on that account, realise and provide instinctively for the special difficulties encountered by blind pupils. He is put into healthy competition with others striving to overcome these difficulties, and the spirit of emulation is fostered. Further, he is set no special number of lessons at so much an hour or boarded out at so much a week. He comes to be trained, and he is kept until he is trained. The period has no fixed limit, for the business of the college or centre is to turn out a healthy, independent, and active citizen.

Again, at St. Dunstan's the social side of life has a full and necessary importance. The blind must not only be taught to be useful independent citizens—they must be taught to play as well as to work. That is a side of training which, until to-day, has never been realised at all, and yet we who work here are daily impressed by the power of enjoyment apparently so largely lost with the destruction of sight, which comes bubbling to the surface as the patient regains the consciousness that the joy of life has only been masked. It is a sight for wonder and a sight of the keenest pleasure to see the men dancing or out on the lake rowing. There is not a suspicion of force or endeavour. One realises at once that it is all the natural outcome of physical spirits once more finding its true way into the life of the blinded man. We have a band here and concerts, and family tea-fights and physical exercises on the lawn, debates on current topics; and, in fact, all the exercises and pleasures of a collegiate life. And the converse side of the question, the teaching the blind to play as a means of teaching the blind to work, is of no mean importance. The old adage of "All work and no play makes Jack a

dull boy" is as true for the blind as for the sighted. Henceforth there is to be no more sitting at the fireside twiddling the thumbs with nothing to do, but a handicraft to pass away some of the tedious hours. What we are beginning to ask ourselves now is—what we cannot teach the blind, not what we can.

All these things, which I have hastily and insufficiently sketched, come from centralising, and can only be done by centralising. That is a point that we have got to drive home. No more overlapping of interests and money. Economy of effort, economy of money, and plenitude of success attend the realisation of this point.

And now, having hastily sketched the first great principle of St. Dunstan's effort, that of centralisation with its attendant advantages to the blind and their training, let me pass on to its corollary—the After-care Department. This great work, as important in its way as the actual training itself, owed its inception to the principle of centralisation. It consists in following up and looking after the blinded man when once more he seeks his place in the world. Such a work as was conceived and is now being carried out could only spring from a centre. Were it not for the After-care Department the blinded man, on completion of training, would be left more or less to shift for himself, or, at the best, dependent on what local effort might by chance be forthcoming to help him over the unavoidable difficulties of his new life. Thus a blind man needs active supervision as regards his work; mistakes creep in, which not being seen are not corrected, and so his chance mistake becomes a habit and the character of his work inevitably deteriorates. So, too, the purchase of suitable materials and the marketing of his goods require active and intelligent help; and, in short, kindly supervision in all sorts of ways is a most necessary factor to help a blinded man to compete on a fairly equitable standard with the sighted man. For this special work a special knowledge of the man, his capabilities, and his capacity are required. All these factors

of knowledge are first gained at his centre of training, and on leaving he is, so to speak, passed on to the After-care Department, which starts its work on his behalf already equipped with all the information that can advantageously be put at its disposal for the future guidance of the man. Time does not permit me further to expand on this part of the work, but the more one sees of the work for the blind the more obvious does it become that training must be followed up by a continuity of perpetual effort. The ideals set out at the beginning of this Hostel have continually and progressively expanded and will continue to expand. The after-care work was a revolutionary conception. Nothing like it has ever before been attempted, but just as the vital importance of it is realised, so too the colossal nature of its task demands that it shall be simplified in so far as is possible by centralising the effort.

There is just one point in this connection which needs a word. There is a natural tendency just now to put our blinded soldiers on a very high pedestal indeed. The pedestal is not of their own choosing, and all of them cannot live up to the height of it. I mean that the men are not the wingless angels to whose ranks for the moment they are exalted, and it is just this that makes the judicious handling by an After-care Department of such special importance at the moment. There is a natural tendency to spoil good men by over-petting, whilst others, who are far removed from righteousness, are correspondingly influenced to take undue advantage of what they can get. All this will remedy itself in time, but at the present it is more than ever desirable that there should be a body to which reference can always be made with regard to a man's antecedents, character, and so forth.

And now, having said a few words on the past and present, I will conclude with a few hopes as to the future.

One great problem of dealing with this new crowd of

comparatively healthy, blind men is the eternal sex question. Now, it is quite certain that the best thing for a young blind man is marriage. It is most advisable from every point of view. The sexual point is so obvious that it needs no further remark, but just as important is the fact that the blind man cannot see how he looks to the world. He is as happy with dirty clothes as brushed ones—he cannot see if his nails are clean or his boots dirty and so forth, for one can go on multiplying the needs of a helpmate almost indefinitely. Unfortunately the blindness of a man brings him into great disadvantage in the marriage market. From the man's point of view he cannot look upon the lady, and so he may fall an easy prey to some worthless virago who would marry and make him miserable. From the woman's point of view, marriage with a blind man means giving up her life to him in a much more complete sense than is usually associated with matrimony. The non-realisation of this plain fact must tend to failure in marriage with a blind man. Thus the question, important as it is, teems with difficulties, as we have already found out at St. Dunstan's. The After-care Department may in the future do very much in protecting the men from unscrupulous women, and may, perhaps, if I may be permitted the gentle hint, be allowed to associate itself with the introduction of eligible maidens desirous of consecrating their lives to help on the good work.

On this account, too, I much hope that in the near future homes or clubs for young blinded men may be formed over the country where, after training, they may live and feed and be looked after, going thence to their daily work. Such homes would be an inestimable boon, and, if properly run, would keep the men from all sorts of temptations to which they might fall an easy prey.

Lastly, we have now to remember that the work being done for blinded soldiers has raised the cry—a somewhat bitter one—from the blinded civilians that all is being done for the soldier and nothing for the civilian. It is true in a sense and for the moment, but it is to be

remembered, as Sir Arthur Pearson said to me when we were talking of this subject, that the blinded soldier is the greatest advertisement that the blinded civilian has ever had. If it had not been for the war, St. Dunstan's would not exist. Without exaggeration it can be said that to-day no war charity has a hold on the people of England to the extent of that one which is given up to the help of the blinded soldier. The treatment and training of the blind has been revolutionised, and the benefits thus obtained for the blinded soldier will inevitably and in due course be handed on to the blinded civilian.

Mr. H. B. Grimsdale :

In this discussion on the question of employment of the blind, it would be absurd for any single speaker to attempt to cover the whole subject. Therefore I have suppressed all extraneous matter and propose to speak to the Society on one point only, that is, on the question of what professional work may be undertaken by the blind ; and in this I use "professional" as opposed to "handicraft."

It is often forgotten that in some important respects the blind professional man is less at a disadvantage than the blind handicraftsman. Yet it is obviously true. The blind man's brain is as good as the brain of a seeing man, and his brain-work is comparatively little hampered by the want of sight. Nay, the necessity of training the memory may place the blind man in an advantageous position in certain cases.

We all know how much can be done by blind professional men ; if even we have forgotten the outstanding example of Henry Fawcett, political economist, statesman, and postmaster-general—political reputations are proverbially of short life—yet we have constantly here before us a monument *ære perennius* of the work of a blind man whose devotion and organising capacity bid

fair to raise all blind men in the country to that position of self-support and self-respect which all who work for the blind wish to see attained.

I assume that the Society wish to hear of remunerative employment, and therefore I shall say nothing of employment which is interesting and recreative solely.

And at the outset I wish to emphasise a truism, so obvious that it seems hardly worth stating, yet it is frequently forgotten.

No one employment can be recommended for all blind. Many factors decide the suitability of any profession for any individual, and if a larger percentage of blind men are able to earn a livelihood by the practice of music than obtains among sighted professional men, this does not mean that all blind men are musical.

It means that among the professions open to the blind, music is that in which they are least handicapped by their disability.

The blind who may be educated for some remunerative occupation may be divided into two main classes, by age. Those who are blind during their school life; those who become blind in early adult life. In normal times the former largely preponderate; during the present war the question of the young adult blind has assumed a special importance, owing to the sudden influx of a large number of wounded soldiers, but this we may hope will be only temporary.

The division is of great practical importance, because the profession of music offers no chance of real success to anyone who begins its study after school age.

Our aim must be to let every blind child have his chance of education to show what he is worth; if his brain is of good quality, do not let it be wasted to the country by forcing him into some badly-paid handicraft, at which he can never compete on an equality with his sighted fellows; let him use for his own and for his country's good his best gifts to their best advantage.

One of the best known workers for the blind said in

his evidence before the Commission: "If a young man or woman has the slightest possibility of earning a living by brain-work, his chances are a hundred to one against the chances of earning a living by his hands. He is handicapped all the time with his hands, but when he comes to working with his brain he is as good as any other man. There is no doubt that he ought to be given that chance."

There are many factors in choosing a profession for the blind child.

The blind child has just as much personal equation as the sighted, and if the choice of professions are limited, the choice must be made with the greatest care.

Blind men have made successes at music, teaching, in the Church, Government (Fawcett), poultry farming, and many varieties of business.

In all handicrafts the blind man is indubitably severely handicapped by his blindness; in some more in some less, but in all it must be a severe handicap.

In professions the handicap is not so marked; the blind man's brain is as bright and his memory is usually better than that of his sighted competitor, therefore it seems obvious that we should encourage any blind man to take up any profession that means brain-work rather than a handicraft, at which, except in most unusual circumstances, he will not be able to compete at anything like equal terms with the sighted.

Now, what professions are there which are open to the blind man, and how can we prepare him for them?

In this short paper I propose to speak only of what I have had experience, and my experience has been gained at the R.N.C. Here, as you all know, the special training is directed largely to the development of any musical faculty, but there are other though subsidiary employments, the chief of which are teaching, typewriting, and last, but not least, the learned professions—law and theology.

To take the last first, the Church has a considerable

number of blind clergy; six or seven, I think, received their education at the Normal College. The life of a clergyman depends less perhaps on his power of sight than that of any other man, but without a strong natural leaning toward it no one should think of entering thereon.

Blind lawyers are not numerous, and I know of one only who was blind when trained; but there are others. In America special grants are made by the States to blind students of the law at the university to pay for the services of a reader; it is not practicable to have all the books necessary printed in Braille. In this country there is no such grant in aid, and therefore the blind man is very disadvantageously placed. He must have sufficient private means to allow him the constant services of a reader to enable him to tackle the subject, even for examination purposes.

Teaching would seem at first to be one of the best openings for the highly-trained and educated blind man. There can be no doubt as to the value of blind teachers in blind schools, and in Great Britain there are a very considerable number of teachers of the blind.

Before the Commission evidence was given by many authorities, and all spoke highly of blind teachers, though it is only right to say that those who supported them most strongly were those who had most experience of their work, and the sighted authorities were not always very enthusiastic. At the same time, the record of the London County Council blind schools, in which from 30 to 50 per cent. of the teachers are blind, shows that they are not unappreciated.

I do not know precisely the number of blind teachers in all the schools for the blind in Great Britain, but in the United States there are 112 among the 449 teachers of the blind. Probably the number is about the same in England.

Mr. Latimer, the head of the Maryland School for the Blind, says that the blind teachers in that school are the

best teachers. The blind teacher thinks in the same terms as the blind scholar, the sighted man thinks in sighted terms and then translates the result into Braille or other blind symbol.

Further, the examples of the blind teacher—his known success—is a great help in encouraging the young blind to follow in his steps. Mr. Latimer says that he has often been told by his pupils of the good his presence has done in that way.

There is, at the Normal College, a branch training school, called the Smith College; the training is long and laborious, lasting at least five years; for many years the candidates have been few; at the time of the Commission it was stated that there were no students then under training. The principal tells me that he discourages by all possible means those students who wish to enter on this career, because time has shown that success in after life is not commensurate with the time taken in training; many of the trained teachers are not able to obtain employment of any kind, and have, after a vain struggle, to give up hope of teaching and begin again to learn a new method of livelihood.

Vacancies are very few among the posts for blind teachers; the principal told me that he did not think there had been more than two or three in any year for a long time.

The blind staff of a school cannot be increased beyond a certain portion; even with the best arrangements it is necessary that the blind teacher's work should be complemented by the work of a sighted teacher, who can overlook the boys in his blind colleague's class in such a way as to ensure that they are not losing the little amenities of life which are taught with difficulty to the blind child; yet the insight which the blind teacher has into the difficulties of the blind child, makes the work of the blind teacher superior to that of the sighted teacher, however sympathetic.

Comparing the statistics of efficiency with those of

music: Teachers, 59; of these 14 are not classed as successful. This is practically 25 per cent. And the results would be yet more unfavourable if some of the recently trained teachers had not been able to obtain employment as typewriters.

Typewriting.—It is not difficult for any blind man to become a fairly proficient user of the typewriter, and at many colleges its use is taught to all the pupils, but primarily as a means of private intercourse rather than as a means of livelihood.

It is amazing to one who is unconnected with this work how rapidly and how easily the blind acquire the typewriter habit. The Normal College pupils make out the most complicated forms and columns of statistics with the greatest accuracy, and, combining the use of the typewriter with a system of shorthand Braille, they are able to take down and copy out on the typewriter as quickly as many stenographers.

But typewriting, as a profession, must always be surrounded with many limitations. The blind cannot read from MS., and therefore there must be someone to translate the MS. into Braille or to read it to the operator. Nor can they correct their own mistakes, though these are remarkably few.

It is, in fact, in the more complicated forms of typewriting, *i. e.* the preparation of tabulated statements, etc., that the inferiority of the blind is least noticeable, and it is certain that in a large office a considerable number of blind writers might be employed on that part of the work which is suitable to them without any sacrifice of effort.

The first question which we will ask on coming to the last part of the subject, viz. on music as a profession, will be the utilitarian one: "Does it pay?" and to that we can give a decided "yes."

Of the 377 pupils who have graduated in some branch of musical education from the R.N.C., about 318 are successful in earning their own living, and some of the

best organists and musicians in the country received their training at the R.N.C.

Somewhere about 85 per cent. of the graduates are earning a livelihood in competition with the sighted; I do not think any other group of professions could show figures approaching this.

The musical training at the R.N.C. is a long one; it begins early, as soon as a child is discovered to be musical, and goes on *pari-passu* with the general education. The child is taught manual dexterity and trained physically all the time that his musical education is proceeding. Most of them learn harmony and counter-point; all learn the mechanism of the piano; tuning is taught with repairing, and lately the pianola has been added as a study.

Great attention is always given to hand training; many of the pupils, when they first come to the College, are unable to dress or feed themselves properly; as the chief object of the training is to make the blind man independent, the necessity of complete mastery of these elementary points is obvious. All children have instruction in class singing; special attention is paid to breathing, voice production, and pronunciation. Instruction on the pianoforte begins usually at seven or eight. All the children have lessons in Sloyd work. For the young children a large amount of the musical work is done in classes, but for the older and more advanced pupils individual tuition is necessary. It is the chief point, perhaps, at the N.C. that the blind musician must have as good teachers as his sighted competitor. Therefore the best teachers have been obtained; further, the position of the College, near the Crystal Palace, has given opportunities to the pupils of hearing, almost every day, concerts of great variety. These have unfortunately ceased at present.

The directors of various musical societies give the pupils of the Normal College privileges, which to some extent counterbalance this loss.

Those who are trained as organists are given oppor-

tunities of choir-training, and are encouraged to act as deputy organist on occasions.

As to the tuners, Sir Francis Campbell was convinced that they must be more carefully prepared even than their sighted competitors; they are all taught to play, they have instruction in voice production and development, they study harmony and counterpoint; and, by daily choir practice, cultivate a good musical taste.

They are taught repairing, and the preliminary tuition in manual dexterity acquired in the junior school, serves them in good stead in the technical work.

Capt. A. W. Ormond

said he would like to elaborate somewhat what Mr. Lawson said about the matrimonial question, especially in regard to training candidates for matrimony. In 1911 there were 45,000 persons who could probably be classified as blind in the United Kingdom. Therefore there seemed to be a large opening for the excess of women who, one was told, existed, and who required work or some form of occupation. And he would suggest to Sir Arthur that it would be an excellent thing if here, at St. Dunstan's, they were not only training blinded men for the purpose of living normal lives, but that there should also be trained women who would be capable of running clubs, such as Mr. Lawson mentioned. He had not previously thought of clubs, but he had thought it would be better if there were women who were properly trained who could cook, read Braille, typewrite, and who understood the general management of a home, or a house, in which lived four or five blinded men who could support the house by their industry. The idea was that in this way there could be established homes for blinded unmarried men. He did not know whether the After-care Committee had already considered this. Perhaps Sir Arthur would think about it, and decide whether it was of any value.

Another point he wished to refer to was, a recommendation made at a meeting of the Section of Ophthalmology of the Royal Society of Medicine, that before individuals were pronounced blind, they should be certified to be so by a properly qualified ophthalmic surgeon. He had a letter from Mr. Stainsley, who was the Secretary-General of the Institute for the Blind, in which he suggested that when a blind man came for a decision as to an occupation, his case should be submitted to a committee, on which there should be an ophthalmic surgeon as well as a physician. This would save the person from entering on a course of life in which there might be a considerable amount of disappointment.

Another point was one which specially concerned ophthalmic surgeons. Sir Arthur was somewhat damping in his remark as to ophthalmic surgeons trying to save even a little sight, but he, the speaker, thought every effort should be put forward to render these men presentable. For a blind man who had to go out into the world to make a living, it was a very great factor in his earning capacity that he should be presentable, *i. e.* not disfigured, or at least, disfigured as little as possible. It was for the ophthalmic surgeon to make an effort to render the disfigurements as small and as unobtrusive as possible. To this end, the man should be encouraged to submit to the fatigue of the needed operations to bring this about. Often a man would put the question : " Can you make me see, doctor ? " and often one had to confess one could not do that, and he had sometimes found it difficult, in those circumstances, to persuade the man what a difference the operation would make to his comfort and his welfare : how necessary it was that the exposed areas of conjunctiva should be covered in, so that he should not be revolting to ordinary people. That aspect would be more clamant after the war was over ; at present it was deemed a meritorious thing, as it showed how much the man had risked for the sake of his country. But later, it would be considered a disability if the blinded man were disfigured.

Mr. Rowan

said he was struck by one case, which showed how a man might continue at his trade, though blind. He was a miner. He told the man he would be blind. He afterwards lost sight of him. He turned up some time after, looking quite tidy, and with a wife, and he told him he was able to earn as much money as other men; that he knew he had to go blind, and had learned to face it. Presumably he worked by touch. He wondered what could be done in the way of getting back to their own trade men who had been wounded in the war.

In conclusion, he would be glad if Sir Arthur Pearson would define, if possible, what the sense of obstacle consisted of: could it be described in any way?

The President

remarked that Sir Arthur Pearson had told the meeting that all things succeeded at St. Dunstan's, and he, the speaker, thought the Society's visit there this afternoon had been a great success. When it was proposed that the Ophthalmological Society should have this discussion on the employment of the blind, some people asked what the Society had to do with that subject; it was even hinted that a doctor was better employed at the bed-side than at a funeral. He, however, did not regard that analogy as quite apt. When a patient died, there was an end to his suffering; when a patient lost his sight some of his worst sufferings were about to begin. Ophthalmologists had sometimes to pass a sentence of permanent loss of sight on a patient; as medical men they had still to treat the patient, and, if possible, endeavour to mitigate the severity of the sentence they had to pass. Their visit to St. Dunstan's that day, and seeing how cheerful people could be who had lost their sight, would, he thought, enable members to mitigate the sentence very materially.

When he saw the sightless men here so cheerful, it made him think there must be some truth in the traditions about Democrates, who was supposed to have put his own eyes out, that he might be able to think more intensely, and who is proverbially known as "The Laughing Philosopher."

Before members left, he would ask them to pass a hearty vote of thanks to Sir Arthur Pearson for his address and permission to visit the institution; he hoped it would be carried by applause.

Capt. CRUISE

said that Capt. Ormond and he had talked over the question of the disfigurement of soldiers with facial injuries, and they agreed that ophthalmic surgeons could not be too patient or too persistent in their efforts to make the subjects of such injuries as presentable as possible by plastic surgery. Sir Arthur Pearson had stated that it was much easier to teach men who were completely blind than to teach those who had a vestige of sight remaining; but that statement must not in any way affect them as ophthalmic surgeons in endeavouring to save or restore even a minimum amount of vision, where there was the remotest possibility of being able to do so. He had one case in his mind, which was very pertinent to the above comment. It was a case in which the man had only one eye, and that was shrivelled and shrunken. All that was left of the cornea measured 4 mm. in diameter. There was a kink in the sclerotic above, the lens seemed to have been dislocated backwards, and caught up in the shrunken scar of the sclerotic. The case looked really desperate, and the man had doubtful perception of light only. He, the speaker, had performed three or four operations, and the man could now see to walk about, and was acting as a pilot to his companions at St. Dunstan's. He had driven the man back only yesterday from the 3rd London General Hospital to

London, and the pleasure of the soldier at again being able to know what was a tree and what was a motor-bus had very intensely impressed upon him, the speaker, a modification of an old adage, "Never say blind."

Sir Arthur Pearson

said, in reply, he desired to thank those present very heartily for their kindness in showing him that they appreciated their visit to-day. He assured them it had been a great pleasure and privilege to him.

He would like to refer to what had been said by Capt. Ormond and Capt. Cruise, both good friends of his. He wished to remove any impression his words might have produced that there could be any but the greatest good in trying to save even slight degrees of sight, provided it were of some use. Mathison had got a useful degree of sight, and if he had sufficient to enable him to find his way about, let him go down on his knees and thank God for it, because it was a priceless possession. What he meant was, that any degree of sight less than enough to enable one to get about was not worth having.

Mr. Arnold Lawson made some interesting remarks about blind men marrying. He thought there was much in what Mr. Lawson said. Marriage bureaux offered a chance, but there were difficulties in that regard, though something was being done on those lines. Girls who came here passed through a good scrutiny, and in only one case had a marriage turned out badly. All the others seemed to be getting along very well. Capt. Ormond's suggestion about training women to look after a coterie of blinded men was very valuable, and he felt indebted to him for it. It would be looked into.

He would like to say a word about the interesting remarks of Mr. Grimsdale with regard to the intellectual employment of blind people. He had very little time in which to address the meeting at the opening, or he would have dwelt at considerable length on the efforts being made

at St. Dunstan's to return men to their original occupations. He thought they had done that with an amazing degree of success, and he hoped there would be many further successes to record. There were several fellows who would go back to their businesses, and he was sure they would acquit themselves as well as was done in the case of two typical instances, one that of an officer and the other a private soldier. The officer was blinded, and, for four months, was a prisoner in Germany, and during that time he had had time to think things out. Before he went out he was a colonial director of a large firm in the City, doing a big export business. He was, when he returned to England, so to speak, very resigned, satisfied to go along as well as he could, but very dubious and uncertain about his future. He had a wife and a small family. He, Sir Arthur, had a long talk with him, and asked him if he was willing to take his advice. The answer was, "Yes." He then advised him on the next morning to go to the city offices of his old firm and report for duty. "What duty?" he said, and the reply given was, "Your old duty." The result, at the end of a long talk, was that he said he would do it. He, the speaker, advised him to think up anything which had transpired when he was there before. If the Johannesburg agent had got into trouble in the earlier days, he could ask him if it had been straightened out yet. This would show that he still had the business in his head. The result was that this gallant gentleman, who had faced his handicap with extraordinary courage, was to-day filling the office of colonial director in the company, and recently, at a directors' meeting, he was confirmed in his appointment, and his fellow directors had told him that they thought he was now doing his work better than before he went to the Front. The officer himself was of the same opinion.

The other case, that of a private soldier, was a man who had been in a good position before he joined the H.A.C. He did not care to follow the suggestions made

as to his future; massage he did not like, and he did not care for shorthand writing. He was formerly attached to a firm of hot-water engineers; he was the member of the staff whose work it had been to fit large buildings with heating apparatus. He, Sir Arthur, asked the man if he had a pretty good general idea of the business, and he said, "Yes; I was in it eight years." He then said to him, "Go back to it." He replied, "But I can't do the work I used to." "Of course not; but you can do other things." He wrote to the head partner, asking him to come and see him, the speaker. He came, and promised to give the man a fair chance. He was taught Braille typing, and put through a course of office instruction and routine. He had read to him the latest books on hot-water engineering, and he was posted up carefully in the recent developments of the business. It was arranged that he should have a girl—whom Sir Arthur picked out—as secretary, and whose wages were to be found for six months. He kept track of that man. In three months this man was doing all the ordinary correspondence of the firm, and was taking his turn with another employee in showing the visitors the latest models of radiators and heating apparatus. Now, two months later, he was doing a thing which he could scarcely ask his audience to believe, though it was quite true. The man who used to do his job now took his plans to him, ran his forefinger over the plans, the blind man then dictated the specifications which the foreman entrusted with the job was to carry out, and ordered the necessary materials. Before he was hit he was being paid £3 a week: his wages now were £4 a week; and he, Sir Arthur, had it from the firm that this would soon be increased to £5, and the man's secretary was paid as well. In the opinion of himself and of those for whom he worked, he was a better man than before he lost his sight.

He could tell the meeting of more cases of the same kind had there been time, and he was glad Dr. Grimsdale had raised the point, because it was, to him, one of the

utmost importance. The first question he asked a blinded man was, "What did you do originally?" and his hope was that he would be told something of his past which would enable the best to be made of his future.

Before the meeting separated, he wished to take the opportunity of thanking three distinguished members of the ophthalmic profession for the valuable help they had given to St. Dunstan's. He wished to offer an expression of the most sincere gratitude on behalf of himself, and, above all, of the men who had benefited, to Mr. Arnold Lawson and Capt. Ormond, through whose hands they all had passed. Capt. Ormond attended to men who went to the 2nd London General Hospital, and Mr. Lawson attended those who came from other camps. Capt. Cruise also placed his services freely at the disposal of blinded officers. He wanted the meeting to understand how grateful all at St. Dunstan's felt to those three gentlemen for the very great help they had given, both in the general working and in their hospital work, in regard to the blinded soldiers.

The sense of obstacle was a very interesting one; he was getting it pretty well now himself. He believed it depended on three factors—echo, difference in air-pressure, and difference in temperature. There was clearly a difference of temperature between a stone wall and the surface of an open paling; one was cold, the other was practically the same temperature as the air. If, in the open air, one was approaching a wall, much would depend, in the matter of perception, on the height of the wall. If it were higher than one's head, its presence could be detected at a greater distance than if it were only two or three feet high. Indoors, on carpeted floors, the question of echo scarcely came in at all; what was most obvious was the difference in air-pressure which was noticeable in still air, as in a house. Change of temperature was also quite definite; most things were a little warmer, or a little colder than the air surrounding them, and there was an almost indefinable change of air-pressure by which one

became conscious of the presence of an object which was not discernible in any other way. The one abiding danger to blind people, in which the sense of obstacle did not apply, was a thin object, such as a door half-way open. That was a horrible danger. One's foot missed it, and the first part of the body to touch it was the face. In his house and here all doors must be shut. Unless one is quite sure about this matter, it was always best to approach a doorway with half-bent arm. There need be no fear of hurting oneself by walking into anything which was fairly wide, because the foot would touch it first, if one were walking upright.

V. CONGENITAL ABNORMALITIES.

1. *An unusual case of opaque nerve-fibres.*

By Leslie Buchanan.

(With Plates II and III.)

The following notes refer to a case in which a small, but interesting and very unusual, congenital anomaly was found in an eye.

The condition may be briefly stated to be congenital opaque nerve-fibres having a course between the retina and the hexagon pigment layer, not, as is usual, in the anterior (fibrous) layer of the retina.

The patient was a man, æt. 45 years, who had an injury to the right eye, on account of which it was deemed advisable to excise the eye. The eye was found to be in an early stage of irido-cyclitis, but there was no extensive inflammatory change. The choroid and retina were congested, and the macula area of the latter was raised in a ridge from the optic nerve entrance to the yellow spot.

The globe had been bisected almost in the horizontal plane and the macular fold remained in the lower half.

A very small, nearly white band of fibres was seen in the optic nerve at its temporal side, and this was noted as opaque nerve-fibres. It was at once noted, however, that the band of medullated fibres passed sharply round the end of the choroid and lay between the retina proper and the hexagon pigment layer.

The utmost radial extent of the band in the eye was about 1 mm., and its breadth might be ·25 mm.

There were lesser bands of medullated fibres, in the

Fig. 1.

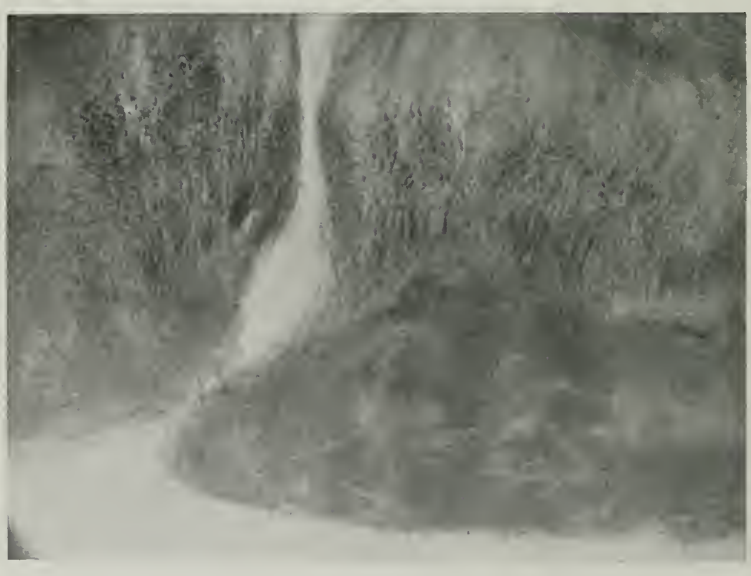

Fig. 2.

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PLATE II.

Figs. 1 and 2 illustrate Mr. Leslie Buchanan's paper on An Unusual Case of Opaque Nerve-fibres (p. 166).

normal position, both to the temporal and nasal side of the nerve.

The optic nerve itself seemed to have a notably oblique position in the sclera, and the abnormal band of fibres took a peculiar double curve round the edge of the choroid.

Sections of the upper half of the globe were made, the lower half being retained as a macroscopic specimen.

Pl. II, fig. 1, shows a section from the central area of the nerve magnified 15 diameters.

The oblique position of the nerve is well seen, and the small band, like a strand of plaited hair, is seen coming up at the temporal side of the nerve. It is notable that the lamina cribrosa is defective or absent at the temporal side, as the strand of medullated fibres is not interrupted at the usual level. Further, it will be seen that, as before mentioned, the band of fibres takes a tortuous course and bends sharply round the edge of the choroid and passes out into the eye at an acute angle. A small portion of the medullated fibres can be seen passing upwards into the fibrous layer of the retina, and this portion is found, in the less central sections, to attain somewhat greater dimensions.

The position of the band of opaque fibres between the retina proper and the hexagon pigment layer, developmentally, between the secondary and the primary optic vesicle elements, is clearly seen. The bacillary layer of the retina is adherent to the inner (anterior) surface of the bundle of fibres.

Pl. II, fig. 2, shows the bacillary layer and its connection with the medullated fibres magnified 150 diameters. The precise nature of this connection is not clear, but seems to be very intimate.

Pl. III, fig. 3, shows the appearances of a very peripheral section of the optic nerve magnified 15 diameters.

The twisted position of the nerve is shown by the unusual sharp shoulders which the nerve has behind the choroid. To the temporal side the choroid overlaps the nerve by nearly half its diameter, and on the nasal

side it overlaps it but slightly. The extraordinary square appearance of the nerve is unlike that of any nerve I have ever seen. The band of fibres in the retina has almost disappeared, and where the unusually sharp and prolonged edge of the choroid comes in contact with the nerve, as it enters the globe, it is seen that the bacillary layer does not reach the nerve-stem but ends some little distance from it. In place of the bacillary layer tapering off, so that the last elements lie close to the nerve-stem, as in normal cases, there is an interval in which appear only ragged ends, of what I take to be nerve-fibres cut in an oblique direction, coming down on to the choroid, which is here but feebly covered by pigment cells.

Pl. III, fig. 4, shows the characteristics of this portion of the nerve head magnified 150 diameters. It is seen that the medullated fibres are diminished to a small bundle, which lies between the ending of the bacillary layer and the termination of the hexagon pigment cells.

Fig. 5,* shows the appearances of the nasal margin of the optic nerve entrance. The lamina cribrosa is seen to be well developed, and is bent somewhat backwards as if it were being crowded into the nerve head. The edge of the choroid is not nearly so sharp or so long as at the temporal side, and the bacillary layer of the retina comes almost to the end of the lamina of Bruch. Beyond the edge of the choroid in the nerve-stem, fibres are seen passing down (backwards) into the nerve, but in place of a direct continuity with the nerve-fibres, as is usual, there is here an interruption. There is a mass of tissue in the nerve, just as it enters the scleral canal, which is quite unusual. This is not clearly defined in its composition in any section, but strongly resembles a mixture of fibres and cells lying irregularly disposed. It is probable that these are retinal elements which have been displaced, and that they have been pushed into the optic nerve because there was no room for them in the normal position.

* Fig. 5 not reproduced. For appearances refer to the nasal margin of Fig. 3, where the characters are clearly shown. – L. B.

PLATE III.

Figs. 3 and 4 illustrate Mr. Leslie Buchanan's paper on An Unusual Case of Opaque Nerve-fibres (p. 166).

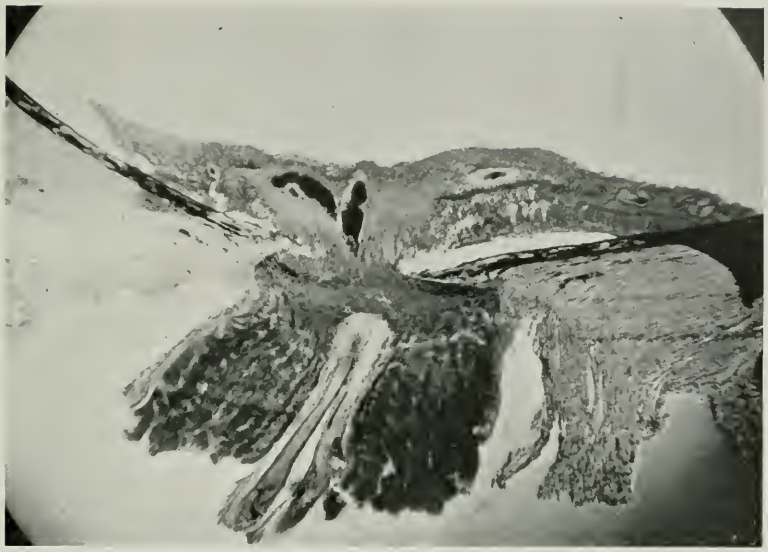

Fig. 3.

Fig. 4.

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Sections of the optic nerve, intermediate in position between these, shown in Fig. 1 and Fig. 3, show a gradual change. In passing from the centre of the nerve to the periphery, the band of medullated fibres on the temporal side gradually diminishes, the portion in the nerve itself disappears first, whilst the intra-ocular portion can be found as noted in Fig. 4. The square-shouldered appearance of the nerve in the optic pore is gradually assumed, and, finally, the mass of tissue in the cup in the nerve, as seen in Fig. 3, only makes its appearance about half way between the centre and the periphery of the nerve.

There are other peculiarities of this nerve head to which I must not call attention owing to time limitations, as I prefer to discuss the more important question as to how a bundle of nerve-fibres, whether they be medullated or not, can find its way into the position in which it is found in this case.

It may be stated here that there was absolutely no abnormality in the patient's other eye.

From a developmental point of view it seems to be almost an impossibility that nerve-fibres of the retina could lie in the space between the primary and the secondary optic vesicles, since the nerve fibres are developed from the ganglion cells. Any theory in explanation of the facts must take account of this point, and also of the fact that the position of the nerve head in the secondary optic vesicle must be more or less fixed by the choroidal fissure.

The only way in which I can explain the occurrence of this very interesting anomaly is by assuming that very early in fœtal life a folding of the secondary optic vesicle had taken place at its posterior pole, and that, by the closing of the choroidal fissure, the retina had been drawn to the temporal side after the development of the nerve-fibres had begun. The result of this would be the curling over on itself of the retina at the part where the choroidal fissure was closing in such a manner that the nerve-fibre

layer, in order to gain access to the optic pore from the displaced portion at the temporal side, would have to pass under the bacillary layer of the normally developing retina at a higher level in the eye.

This theory would explain the absence of the outer layers of the retina at the temporal side of the nerve, and also the presence of the retinal tissue in the nerve head. It would also account for the peculiar oblique position of the nerve itself in its course through the sclera, and the fact that the bundle of nerve-fibres from behind the retina lies outside, to some extent at all events, the normal line of the optic nerve.

As the lamina of Bruch is present in all sections up to the edge of the nerve head, it is to be supposed that there was no coloboma of the choroid or optic nerve.

The PRESIDENT said this was a very unique case. To have opaque nerve-fibres on the outer side of the retina was very extraordinary and very difficult to explain. He thought, however, the case belonged to a class in which, apparently, the outer layer of the optic vesicle had simulated the inner layer. He reported one such case before the Society some years ago, and a drawing of it he now exhibited.* The late Mr. Coats† had also described such cases, in some of which retinal elements had been found in the optic nerve.

Mr. M. S. MAYOU said he thought congenital defects in the retina were much more likely to occur near the disc than elsewhere. He had a specimen of a microphthalmic eye in which the retina was normal except just at the margin of the disc; in that position there was a rosette formation, substituting the pigment cell layer, very much like the rosettes seen in glioma. He agreed with the President that it was much more likely to be the outer pigment layer taking on the characters of the inner layer.

* *Trans. Ophth. Soc.*, vol. xiii, Plate IV.

† *Roy. Lond. Ophth. Hosp. Reps.*, vol xvii, p. 214.

2. *Small tortuous veins on the optic disc in association with small vascular nævi of the skin of the eyelid and of the nasal septum.*

By E. Treacher Collins.

Henry P—, æt. 57 years, has been in attendance at Moorfields Hospital since April 3rd, 1911. He came first for a change of glasses, and later, in 1911, on account of an injury to the right eye from a piece of wood. This injury necessitated the removal of a prolapsed iris; previous to the injury the right eye was, except for a slight degree of myopia, quite normal. The vision of his left eye with $-$ 2 D. is equal to $\frac{6}{9}$. The iris in it is a hazel colour, with a large dark brown area in its lower part. There is a dotted brown opacity in the centre of the anterior capsule of the lens. In the centre of the vitreous there is a linear, stationary, grey opacity running antero-posteriorly, but unconnected with either the optic disc or the posterior capsule of the lens.

The main retinal vessels run a straight and normal course. On the optic disc, and nearly confined to its area, are several small, and remarkably tortuous, veins. Those on the temporal side extend for a short distance beyond its margin into the retina (Fig. 11).

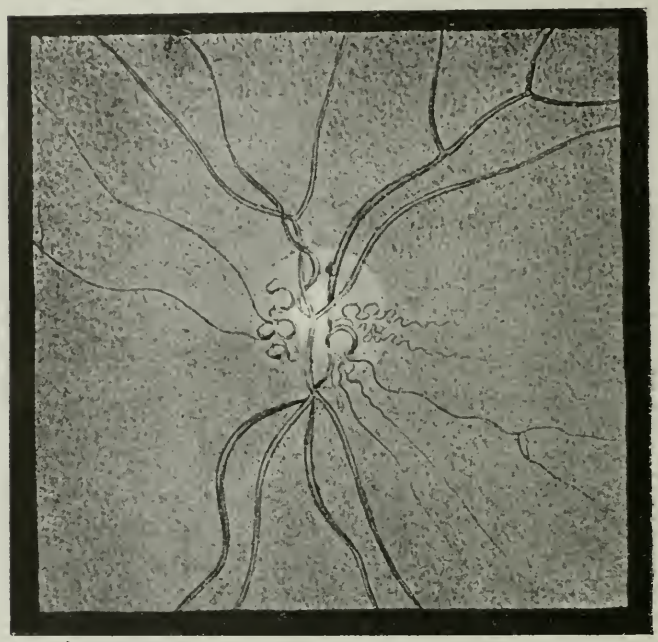
In the skin of the patient's left lower lid are two small, widely separated, vascular nævi. Each is about the size of a large pin's head. In the skin of the left temporal region there is a large, mottled, capillary vascular nævus.

The patient states that he has, since a boy, been liable to bleeding from the nose when he blows it, and that the blood comes mostly from the left nostril. On examination of the nose, a small patch of dilate veins is seen on the left side of the septum.

Remarks.—Cases of tortuosity of the retinal vessels in association with vascular nævi of the skin of the face have been shown at this Society by P. Horrocks, vol. iii,

1883, p. 106; Work Dodd, vol. xxi, 1901, p. 82; and by G. Hartridge, vol. xxi, 1901, p. 83. In the first two cases the main retinal vessels were involved; the appearances in them were, therefore, very different from this case.

Fig. 11.



Small tortuous veins on the optic disc in association with small vascular nævi of the skin of the eyelid and of the nasal septum.

Of Hartridge's case there is no drawing, but he describes it as having a nævoid condition on the disc affecting chiefly the veins. The association with bleeding from the nose and a nævus of the nasal septum has not, I think, before been noted; it may throw some light on cases of hæmorrhage into the eye in association with nose bleeding.

The opacities on the anterior capsule of the lens, and in the vitreous, are doubtless remnants of the fœtal vascular

system of those structures which have failed to completely disappear.

Mr. E. T. Collins said he thought the great interest of the case was the association of these abnormal retinal vessels with an abnormal condition of the vessels of eyelid and of the nasal septum. This man had had attacks of bleeding from his nose. He thought the attacks were undoubtedly to be explained by the rupture of some of these small nævoid vessels in the nasal septum. He suggested that in all cases where attacks of nasal bleeding were associated with hæmorrhage in the eye, it would be well to look into the nose to see if there was a similar nævoid condition of vessels. He had recently seen another case, somewhat similar to this, in which there was a solid-looking mass in the lower part of the fundus with markedly dilated and tortuous retinal veins. It had been suggested that the man had a flat sarcoma of the choroid, and that it would be better to have the eye removed. It had also been suggested that he had what was known as "Coats' disease." The appearances suggested, he thought, those of a case of angioma of the retina, of which there were several recorded instances in the *Transactions* of the Society. The first one, recorded by Mr. D. J. Wood, of Cape Town. As confirmatory evidence that it was an angioma of the retinal vessels, and not a sarcoma of the choroid, was the presence of several small angiomata in the skin of the lower eye, similar to those in the patient shown.

Lieut.-Col. Elliot remarked that he had nothing additional to say about the case. He was much indebted to the President for his help in the above case. The moment he saw the female patient he recognised the character of the case; the case was so much like his own one. An interesting point about the young lieutenant whom the President referred to was, that the detachment increased very much while he was under observation. He had recently been invalided.

3. *Unilateral melanosis of a segment of the iris combined with pigmented nævus of the lid margin and pigmented nævi in the skin of the face in a dark-complexioned subject.*

By A. C. Hudson.

D. G—, male, æt. 7 years. A portion of the lower quadrant of the left iris, comprising about one-half of its extent, exhibits uniform dark brown pigmentation with nodular prominences systematically distributed over its surface, the condition being similar to that described by G. Coats in *Transactions of the Ophthalmological Society of the United Kingdom*, vol. xxxii, p. 165. The uveal fringe of the corresponding pupillary margin is somewhat hypertrophied, but the eye shows no other pigment anomaly. At the posterior border of the margin of the lower lid is a small pigmented swelling of nævoid character, and there are numerous small pigmented nævi in the skin of the face. The eye is amblyopic from squint.

The President: Some similar cases to the one shown were recorded in the *Transactions*: one by Mr. Coats, who compared the elevations on the deeply-pigmented iris to those seen on the surface of a tennis ball. He, the President, showed, at the same meeting, a similar case, in which the elevations looked like those on an india-rubber sponge; that case also had a deeply-pigmented iris. In Mr. Hudson's case the elevations were confined to the segment of iris, which were deeply pigmented, and were similar in appearance to those in Mr. Coats' case.

4. *Congenital buphthalmos, with enlargement of the arteries of the retina.*

By H. Grimsdale.

E. J. P—, æt. 14 years, pupil at the Royal Normal College for the Blind. Has always had large eyes and very defective vision. Has attended various hospitals in Lancashire. Recently came to the Normal College.

V. : fingers badly. In both eyes there is a deep cupping of the optic disc; in the right, the retinal arteries are much larger than the veins, and are tortuous. They have a broad, light streak, and look flattened. The veins are almost normal. The condition of the arteries is somewhat similar to that of the cases of "retinal angioma," but I have not found any mass to which the vessels run.

Mr. Grimsdale said that he regarded this case as allied to the cases of angiomata, but he confessed that he had never seen a similar case.

Mr. Clegg said that the case put him in mind of a case he had been watching, which had very enormous arteries coming from the disc, he thought only in one eye. The artery sprang from the disc to the temporal side of the retina, and one could trace it back to the optic disc in the form of a loop with great sinuous curves. But the cases were not quite similar.

VI. INJURIES.

1. *Protection of the eyes in warfare.*

By R. R. Cruise.

Throughout the Universe, the wits and ingenuity of a large proportion of mankind have been exercised for the past two and a half years on processes of physical destruction and physical protection.

In the latter category, we have seen the ploughing machine evolved into the Tank ; but the ploughman has not yet reached an equal condition of invulnerability.

And the reason is obvious, for wherever the question of body armour arises, the two discordant factors of efficacy and practicability have to be faced.

The steel helmet was the first great and effective compromise between these two factors, and even then, it required a rigid discipline to enforce its usage upon a reckless and reluctant soldiery.

The measure of protection against injuries to the head afforded by the steel helmet will, it is to be hoped, find a parallel in the case of the eye, in the visor which I have designed, which the War Office has accepted and adopted, and which is now on its trial on an extensive scale.

But, before describing the visor, it is necessary to consider the types of injuries to which eyes are liable in warfare.

From the point of view of protection, eye injuries may be divided into two classes :

(a) The preventable.

(b) The non-preventable.

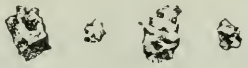
The simplest instances are, of (a) a small shell splinter

penetrating the globe; of (B) a bullet traversing both orbits.

The latter class needs no further attention. The former class demands the most earnest consideration by the ophthalmic world and the military authorities.

To obtain an accurate conception of the extent of this class, one requires statistics covering a large number of cases examined carefully and critically within a brief lapse of time since the injury. Facial wounds heal rapidly, and evidence quickly disappears, so that, even under the most favourable circumstances, there must always be a large category of injuries where the verdict between preventable and non-preventable must remain non-proven.

Fig. 12.



But still there will be a large class where the evidence is clear as to the preventable nature of the damage. It would be wasting your time to elaborate these injuries, so I will mention but a few.

Cases where the cornea is perforated the iris prolapses, and a septic panophthalmitis results.

Cases where a foreign body has had sufficient energy to penetrate the globe, and remain there.

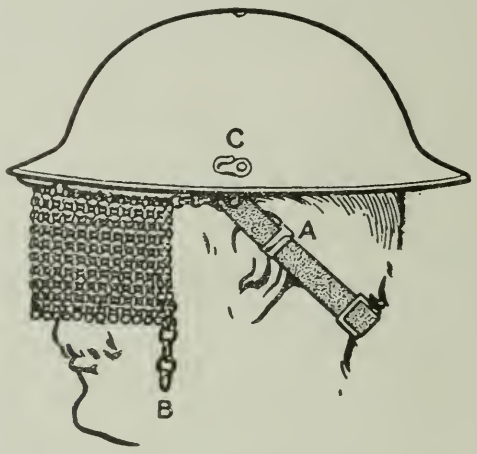
Contusions of the globe, producing a varied and common picture of dislocated lens, traumatic cataract, iridodialysis, intra-ocular hæmorrhage, and retinal lesions; in this respect, I would say, by way of parenthesis, that I have been much surprised by the relatively few cases of detachment of the retina that have occurred in my experience of war injuries.

These, and many similar injuries, are the result of any form of explosive—shell, grenade, or bomb—sending every form of matter—mineral, vegetable, and animal—into the eyes in a shower of particles of every size and shape.

The majority of the injuries caused by this type of casualty are due to small particles.

Messieurs Morax and Moreau, working at the Lariboisiere Hospital in Paris, estimated that 54 per cent. of the casualties to eyes in a series of 486 case of injuries by shell, grenade, or bomb, were caused by *small* particles; and, as the result of a critical examination of 698 cases, conclude that 50 per cent., or one-half of all injuries, could be prevented by some sort of gauze shield (*Ann. d'Oculistique*, August, 1916).

Fig. 13.



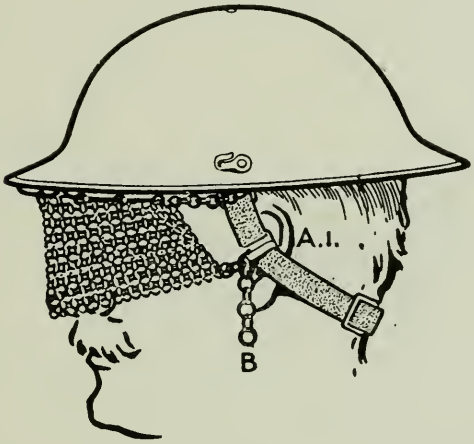
My own experience, and that of others to whom I have spoken, puts the percentage higher; but even if one estimates that 50 per cent. of all eye casualties are caused by small particles, and that there is a practical form of protection against them, it will be admitted that an imperative duty lies before us to insist on expediting the supply of a practical form of ocular protection to our Army.

I do not know, Mr. President, whether you would consider that it was outstepping the province of this Society to send a resolution to the Secretary of the War Office expressing, in the name of the members of this, the

premier Ophthalmic Society, the urgent necessity of expediting the measures adopted against preventable blindness.

My ophthalmic resident, Capt. W. L. Holyoak, has assisted me in examining a series of cases at the 3rd London General Hospital, Wandsworth, and at St. Dunstan's, where, by the courtesy of that indefatigable worker on behalf of the blind—Sir Arthur Pearson—we examined 100 cases, or 200 eyes.

Fig. 14.



The question to decide was what we should consider preventable.

We took a deliberately modest view of the stopping power of the visor of chain mail. We excluded as non-preventable:

All injuries by bullets.

Injuries by shell fragments of sufficient strength to fracture any bones in the orbital, nasal, or malar region.

Injuries producing deep scarring of the face in the vicinity of the eye.

Injuries producing extensive scarring of intra-orbital contents, as evidenced by shrinking sockets.

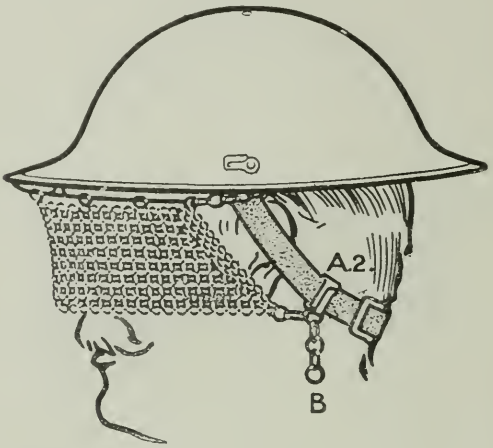
Severe concussion, where the globe was outwardly uninjured.

We classified as preventable :

Cases where the history and appearance made it reasonable to suppose that the injury was caused by a small particle of low energy, causing loss of sight by perforation, or laceration of the globe.

Corroborative evidence was afforded by slight superficial skin injuries in the neighbourhood.

Fig. 15.



We classified as doubtful 16 cases ; but we finally decided to include all these among the non-preventable.

Even so, the percentage worked out :

Preventable, 52 per cent.

Non-preventable, 48 per cent.

These figures are for 200 consecutive eyes at St. Dunstan's only, and are remarkable when one considers that they are figures taken from an institution to which only the most serious cases are sent, involving loss of sight in both eyes.

When we came to examine the figures taken from the 3rd London General Hospital, at Wandsworth, the percentage of preventable injuries was still higher.

All the cases were of comparatively recent date, and the evidence to form a decision upon was more definite, and probably the conclusions more accurate.

As, however, we have only recently begun to consider percentages, our numbers are insufficient for a final statement; but we propose at a later date to give fuller figures when further evidence has been collected.

We have examined 120 eyes in the wards at present, and classify them as follows:

Fig. 16.

Preventable, 65 per cent.

Non-preventable, 35 per cent.

Added to the figures obtained at St. Dunstan's, this yields a total of 320 eyes examined, with the result:

Preventable, 58 per cent.

Non-preventable, 42 per cent.

So that, if a reduced estimate is made that 50 per cent. of all eye injuries are preventable by a protection such as that afforded by the visor, that estimate would accord with the figures and opinions of Messieurs Morax and Moreau, quoted before; and, in our opinion, would be

an under-estimation rather than an exaggeration of the facts.

The visor, as you see, consists of a transparent mesh of steel rings, so closely linked together that only the most minute particle could pass through its meshes, and then only if it struck it at right angles to the plane of its surface ; a tangential blow would meet with a practically continuous barrier of steel.

The visor is attached to the helmet, and is, therefore,

Fig. 17.

not a separate piece of equipment to be remembered and attached to an already overloaded body.

When not required, the visor can be thrown up on to the brim of the helmet, and lowered again as occasion requires by the simple process of unhooking a chain and refastening to another hook.

A chemical process renders the links rust-proof.

If the extensive trial to which the visor is now being subjected in the Army proves as satisfactory as it is anticipated, a considerable stride will have been taken in the problem of preventable blindness.

The PRESIDENT said he was sure the Society would wish to congratulate Capt. Cruise on his invention, and, he thought, the British Army also. He would like to know whether the visor had been put to a practical test on the field of battle.

Sir GEORGE BERRY said, on the face of it, the invention seemed to be a very valuable one. It was, however, possible to conceive that projectile fragments which might otherwise destroy only one eye, might, if the soldier were wearing such a visor as this, do further damage through striking the visor. But even if that were the case, he thought such cases would be more than counterbalanced by the great number in which the protection which it afforded was likely to be useful. But, as the President had indicated, it was first necessary to know how far its usefulness had been demonstrated.

Mr. HARRISON BUTLER said he could see even better through the visor than without it, having 2 dioptres of myopia.

Lieut.-Col. ELLIOT asked whether Capt. Cruise had made any investigation as to whether forward observing-officers had been able to work with the visor, and whether, in the case of these men, who sat for hours watching the enemy in the front line, a visor could be made with small apertures for the pupils. There are a very large number of young R.F.A. officers doing their work along the whole length of our line, and they are very heavily shelled. They must have first-class sight and he feared the mesh of the visor might to some extent interfere with this. Of the great value of Capt. Cruise's idea there could, he believed, be no doubt.

Mr. J. R. LUNN said the helmet and visor combined seemed heavy for a hot climate; he would like to know what the weight was.

Mr. PRIESTLEY SMITH said he thought sufficient evidence had been put before them to justify the Society in calling the attention of the authorities to the matter, with the expression of the hope that the visor might be rapidly made and supplied as widely as possible. He proposed:

"The Ophthalmological Society of the United Kingdom, having examined the steel visor devised by Capt. Cruise, has good reason to believe that it would prevent at least 50 per cent. of all eye injuries received in warfare.

"The visor has already received the strong approval of ophthalmic surgeons in the field and at home.

"This Society respectfully begs to urge that the production of the visor should be expedited, and that its application and method of use should be placed under the supervision and control of a competent expert authority."

Lieut.-Col. Elliot seconded, and it was carried unanimously.

Capt. Cruise, in reply, said the story concerning the visor was an official one, and it would be outstepping his province to give details of it to a meeting such as this. There were many initial difficulties that had to be overcome, but there was room for much improvement in the production and management of the invention, and he hoped to get the matter thrashed out very shortly.

In answer to the President and Sir George Berry, certain experiments had been carried out to test the stopping power of the visor. They had not been very extensive, because the principle was chiefly clinical—by that, he meant that a projectile which had only just sufficient penetrating power to perforate the ocular tunics, and remain in the globe, or cause an incision of the globe, or bespatter the cornea with small particles, must of necessity be stopped by the visor of steel mesh. Recently he had shot at the visor with a choke bore shot-gun, and a full charge of number ·5 shot, at a distance of 35 paces. The visor stopped some of the shot, and some of the links were knocked loose. Then he got half a dozen ox-eyes at the same distance, with the orbital fat and muscles attached, and placed them on a layer of cotton-wool, about 1½ in. thick, and slung them on a deal board, and shot at them. The pellets went through the eye, through the

muscles at the back of the eye, through the cotton-wool, and penetrated the deal board for about 2 mm. Then an eye was shot at with the visor in front, at the same range, paper being placed in front of the visor to indicate how many pellets had struck it. The most interesting pattern was that in which six pellets struck the visor; four were completely stopped, one knocked a link loose on to the globe, and one just penetrated the globe, and remained in it. So that a projectile of sufficient force to penetrate the globe completely, and probably kill the man by penetrating the orbital walls, was completely stopped in four out of six instances, and in only one out of six caused penetration of the globe. Some friends of his had exploded a Mills grenade at a distance of 10 ft. away; the visor stopped all the small particles, and again only some of the links were loosened.

In reply to Col. Elliot, he thought that probably some form of goggle would be better for forward observing-officers. The forward observing-officer was sitting with field-glasses most of the time in front of his eyes, and this would probably afford sufficient protection in case of a shell exploding somewhere near.

The weight of the visor itself was 5 oz. He had, of course, nothing to do with the helmet to which it was attached. The helmet was, of course, a heavy article, made of manganese steel and extremely tough, and he understood, in the opinion of experts, it was the best helmet in all the armies. At present, the whole of the evidence, and the whole of the theories with regard to the visor rested on his own personal statement, and the reports of friends. Col. Lister warmly approved of it, and he, the speaker, had shown it to all the army commanders on the Western Front, to all officials at the War Office at home, and at General Headquarters abroad, and he had received numerous unofficial appreciations as to its efficiency in preventing the type of injury for which it was intended; and that type of injury constituted at least 50 per cent. of the total of all eye injuries in this war. A

shot, which at 35 paces would kill a man from a shot-gun if it struck him in the eye, would, with the visor in position, only penetrate the cornea.

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2. *Pulsating exophthalmos as the result of a wound of the head.*

By M. S. Mayou.

The patient was a soldier and was wounded in France in the beginning of 1916 by shrapnel. The wound of entry is just behind the right mastoid process, and the piece of shrapnel, about the size of a shilling, has been located behind the left coronoid process of the temporal bone. The right orbit was explored in France, and since that date the right internal carotid had been tied in London Hospital. At the present time he has right facial paralysis; proptosis of the right eye and pulsation can be felt in it by pressing the globe backwards into the orbit. There is limitation of movement of the right eye and dilatation of the ciliary vessels. There is a bruit to be heard over the head and orbit. He has corneal nebulæ in both eyes, and his vision is: R. $-$ 2, $\frac{6}{36}$, L. $-$ 1, $\frac{6}{12}$. There is no optic neuritis or dilatation of vessels of either fundus. The case is probably one of arterio-venous aneurysm and does not seem to have been improved by the ligature of internal carotid.

Mr. Clegg had had a case something like this. The carotid was tied and without any result. The man brought an action under the Workmen's Compensation Act, which was settled for a large sum of money, and a year later the eye had gone completely back. He would therefore be inclined to leave the case for several months, at any rate, before resorting to surgical measures. The man in the case he had been speaking of did not complain of noise.

Mr. Elliot said that he had had a case of pulsating

exophthalmos in which the *bruit* was heard all over the head. He was anxious to get surgery to bear upon it, but the man would not consent. He heard subsequently that the case had got very much better, without any treatment.

Mr. Ridley said that he had twice tied the common carotid. One was a traumatic case, the other due to a gumma. In the traumatic case the first symptom relieved was the noise in the head, and the patient was quite well in a few weeks. But in the second case, apparently, no result was obtained. He had mentioned it at the Society some time previously. In this case, long after ligation of the common carotid, the facial artery dilated, until it became as thick as one's little finger, where it joined the orbital arteries. It was tied. The proptosis and the pulsation all ceased, and that patient, although she lost her vision under pressure, had no trouble and practically no proptosis now.

The President said that he had had a patient with pulsating exophthalmos who complained bitterly of the buzzing noise in his head, and not only did he complain of the noise, but his wife also! His carotid artery was tied, and for a time there was perfect relief. The buzzing, however, after a few months returned, but not sufficiently to again annoy the man's wife.

Mr. Whitehead said that in cases where he had tied the common carotid the proptosis subsided and the bruit ceased. Two of the cases were traumatic in nature and one syphilitic. In the case before the meeting pressure on the carotid did not arrest the bruit, and he doubted whether tying the common carotid now would do any good.

VII. DISEASES OF THE EYELID AND ORBIT.

1. *Case of functional blepharospasm in an old man, causing temporary loss of sight.*

By I. L. Tuckett, M.D.Cantab.

T. C—, æt. 65 years, attended at Mr. A. C. Hudson's clinic on April 26th, 1917, complaining of inability to open his eyelids when once they were shut, unless he helped to raise the upper lids by pulling the skin of his eyebrows upwards by hand.

His occupation used to be that of steeplejack in connection with building factory chimneys, though for some years he has not been up a chimney himself but has only helped his son to do the steeplejack work by standing at the bottom of the chimney looking upwards for signals indicating what tool or other material his son required sent up to the top of the chimney.

About the beginning of January, 1916, he began to find this looking-up work very trying, and after May, 1916, (when one day he had a fright, thinking his son was going to fall as he watched him swaying about on the cradle), he found great difficulty in keeping his eyes open to look anyone straight in the face.

He went to the Birmingham Eye Hospital on August 4th, 1916, where he was advised that, as nothing was wrong with his eyes, he had better go to a general hospital—advice which he never carried out.

Present state.—Both eyes are apparently healthy, with 1 D. of hypermetropia.

The fundi are normal.

There is no defect of the ocular movements, or of the levatores palpebrarum when the eyes are open.

When his eyes are open, he stares in front of him as long as he can without blinking; but, as soon as he blinks, the levator palpebræ cannot overcome the orbicularis sufficiently for the pupil to be uncovered, although some of the sclerotic below the cornea is exposed. If now one upper eyelid is raised by hand, the other eye opens naturally.

He can also open his eyes at any time by carrying out the following device: If he pinches the end of his nose between his finger and thumb so as to concentrate his attention on that point, his eyes roll down and in and he begins to see something. Then he can gradually look up straight in front of him and keep his eyelids wide open till the next time he has to blink.

He has a marked tremor of his hands at rest, and his hand grasp is a little weak, but his knee-jerks are normal.

He also says that sometimes, when he is brushing his boots, his hand stops moving and he can't go on brushing the boot.

At present it is not possible to say for certain what is the nature of his blepharospasm, though a psychical cause is certainly suggested by the history.

He is being transferred to St. Thomas's Hospital for further investigation of his nervous system and electrical reactions.

Mr. Tuckett said that there was really not very much to add with regard to this case. It would perhaps be more suitably treated at Queen Square than at the Ophthalmic Hospital, for there were analogous conditions in the hands. Sometimes when the man was brushing his boots, for instance, he could not arrest the movement.

2. *Papilloma of the conjunctiva.*

By M. L. Hepburn.

(With Plate IV, fig. 1.)

A. C—, æt. 7½ years. In August, 1916, first noticed "something on the inner side of the left eyelid." Lotion was prescribed.

In December, 1916, the swelling had grown so large that removal was attempted by operation. The growth recurred very rapidly, and eight weeks later a further operation was performed followed by a similar result.

He came to the Royal London Ophthalmic Hospital on March 29th, 1917, and was admitted. A piece of the growth was removed and examined microscopically, when it proved to be a rapidly growing papilloma, with no evidence of tubercle in the sections.

Mr. Mayou said that the appearance seemed to suggest that the tumour came originally from the caruncle, as it had that leaflike form which papilloma of the caruncle generally have. Whether it had undergone any malignant change he would not like to say, but these papillomata would sometimes spread in this way, rather like the villous tumours of the bladder, without necessarily being malignant.

Mr. Whithead suggested that diathermy might be tried. He had used it for several extensive rodent ulcer cases. It was rather a difficult apparatus to control, and he did not know whether members had any experience of it. He now used a holder much smaller than when the instrument was first introduced, but the tremendous voltage of the current employed necessitated the use of an extraordinarily thick handle and connecting coil; but with a little care it could be utilised, and, following the ordinary procedure of keeping close contact (no breaking of contact must be allowed for an instant, otherwise there would be

PLATE IV.

FIG. 1 illustrates Mr. M. L. Hepburn's paper on Papilloma of the Conjunctiva (p. 190).

A large papillomatous growth occupies the region of the inner canthus, originating from the conjunctiva lining the inner half of both the upper and the lower lid of the left eye. The larger part projects between the lids and, on emerging, expands to form a mushroom-shaped growth resting on the skin between the inner canthus and the bridge of he nose.

FIGS. 2 and 3 illustrate Mr. M. S. Mayou's paper on Two Cases of Herpes Zoster affecting the Ciliary Nerves (p. 226).

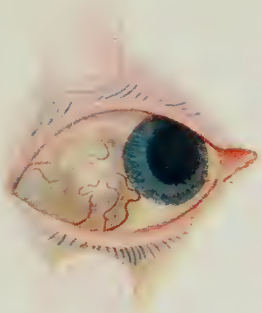

Fig. 2.

Fig. 3.

Fig. 1.

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tremendous sparking), it simply slowly burned away without any heating of the surrounding parts, and there was an extremely small amount of cicatrisation. He thought it a very valuable procedure for rodent ulcer.

Mr. MACKAY said that radium should certainly be tried.

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3. *Orbital suppuration.*

By M. S. MAYOU.

ABSCESSES in the orbit from a practical point of view are often very difficult to find. The object of this paper is not so much to examine the cause, but rather the situation in which pus is likely to occur and "point," and the best means of opening and draining such abscesses. The main object of the paper is to put on record some rare cases of orbital suppuration and some unusual situations in which pointing of an abscess has occurred. For the study of the track taken by pus in the orbit forward to the skin which necessarily takes the line of least resistance, it is important to study the fibro-cellular tissue planes in the orbit. These fibro-cellular tissue planes are arranged in two directions (1) those closing in the anterior wall of the orbit, and (2) those which take an antro-posterior direction. The orbit being almost entirely enclosed by bony tissue, except on its anterior wall, an abscess which occurs within it will manifest itself by a protuberance of its anterior wall, and pus will be directed forwards to it by the cellular tissue planes arranged in the antro-posterior direction. This anterior wall in normal conditions is closed by the fibrous attachment of the lids to the orbit, which is thickest at the outer and inner canthus, where they receive special names of the tarsal ligaments, and thinnest at the upper and inner part near the attachment of the pulley of the superior oblique (Fig. 18). Another plane of cellular tissue which helps to form the barrier to the passage of pus forwards from the orbit is the attachment of the check ligaments. In

these planes of cellular tissue there are three compartments: one contains the lachrymal gland, another contains the lachrymal sac, the third, the eye (tenon capsule). These are all comparatively superficial, and if pus forms in either of these it practically always bursts through the skin or conjunctiva rather than into the orbit.

The connective-tissue planes which are arranged in the antro-posterior direction are the periosteum of the orbit,

Fig. 18.

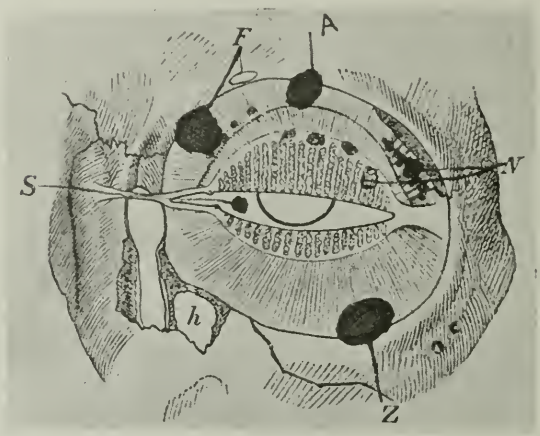


Diagram showing the connective-tissue membranes closing the anterior wall of the orbit (after Fuch), and the usual situation for the pointing of abscesses. A. Frontal sinus. F. Ethmoidal sinus. Z. Antrum. N. Lacrymal gland. S. Tenon's capsule.

and the sheath of the optic nerve. These membranes are all of sufficient density in the early stages to completely stop the passage of pus from one compartment to the other; that is to say, abscesses may form sub-periosteally within the fatty tissue of the orbit or in the sheath of the optic nerve. It is important to recognise this early, as the abscess can be opened before other parts of the orbit are implicated.

Necrosis of the orbital wall may give rise to the for-

mation of a subperiosteal abscess (Fig. 19). The orbit on its upper, inner, and inferior wall is surrounded by the sinuses of the nose in which suppuration may occur giving rise to the same condition. These abscesses as a rule

Fig. 19.

Suppurative periostitis of the frontal bone.

point in very definite positions. Those arising from the frontal sinus usually present opposite the centre of the upper orbital margin. The one arising from the ethmoid presents at the upper and inner angle of the orbit in the region of the attachment of the pulley of the superior oblique (Fig. 21), whilst one from the antrum which is comparatively rare presents to the lower and outer orbital margin. In opening abscesses which arise from necrosis of the orbital walls, it is especially necessary to recognise that

they are at any rate in the first instance subperiosteal, because they can be evacuated without opening the cellular planes of the orbital tissues by cutting straight down on to the orbital margin and stripping up the peri-

FIG. 20.

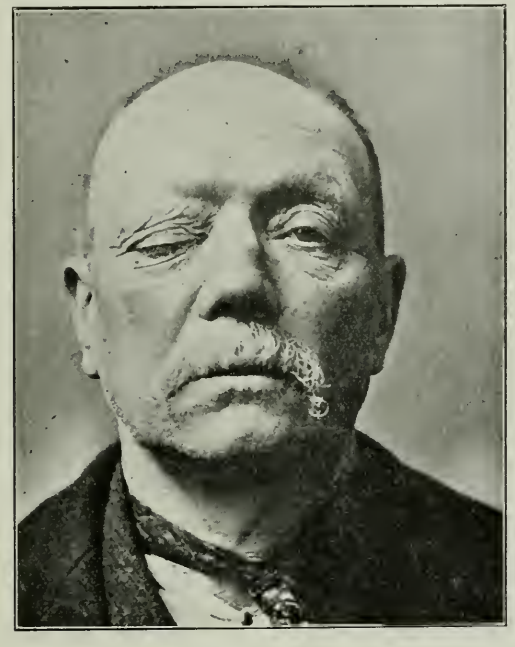
Orbital cellulitis showing bulging of the whole anterior wall of the orbit.

ostium with a periostium detacher until the abscess is opened. In the early stages of the condition it may be possible in some instances, especially in the case of the ethmoid, to open the subperiosteal abscess from the nose after removing large portions of the ethmoid (Fig. 21).

In what I believe to be an almost unique case, reported below, infection of the ethmoid arose from a boil on the face associated with a subperiosteal abscess (Case 1, Figs. 23 and 24).

Abscesses which occur in the fatty cellular tissues of the orbit are most frequently the result of a penetrating wound, cellulitis of the face, or pyæmic infection, and at first give rise to diffuse infiltration with a general swelling of all

FIG. 21.



Suppuration in the ethmoid presenting at the upper and inner angle of the orbit; the abscess was opened by Sir StClair Thomson through the nose.

the orbital tissues and later may point in any position, more commonly in the upper part of the orbit, as in that position the fibro-cellular tissue is thinner than elsewhere (Fig. 20). The difficulty in treating such abscesses is to find them, if not pointing, and the best method is after incising the lid to search for them by means of sinus forceps. When opened and drained pockets of pus are very

liable to form in the orbital fat and drainage taking place imperfectly.

Abscesses in Tenon's capsule are nearly always pyæmic in origin, although it is conceivable they may result from a perforating injury or a tenotomy wound, or possibly as

FIG. 22.

An abscess in the deeper tissues of the eyelid simulating an acute frontal sinus.

the result of panophthalmitis. As a rule the abscess which is formed bursts forward beneath the conjunctiva between the insertion of the recti muscles, but occasionally perforation of the globe may take place as in the case reported by Bronner (*Trans. Ophth. Soc.*, vol. xxiv, p. 209).

The close contiguity of the pus with the eyeball produces acute inflammation in the uveal tract with the

exudation of leucocytes into the vitreous. In the first case described below the pus in the vitreous contained no organisms although the pus in the cavity of the abscess contained staphylococcus, so that it is probable

FIG. 23.

Suppuration in the ethmoid, right side orbital cellulitis, on the left infection from a boil on the bridge of the nose.

that the exudation into the eye is the result of the toxine rather than the true infection of the eyeball with the organism. This is further borne out by the other case, as after the abscess had burst through the conjunctiva the eye, which contained a yellow mass of leucocytes in the vitreous, gradually settled down, and the eye was retained by the patient, although it showed some signs of shrinking at a later date. This inflammation within the globe is, I think, very important in making the diagnosis in the early

stages of the disease, as it is conceivably possible that the abscess might be opened through the conjunctiva before the eye is destroyed. Abscesses of the optic nerve are of extreme rarity. Cases have been recorded as the result of infection from the face or spreading from the meninges.

Fig. 24.

Same as last photograph, showing recovery.

Case 1.—*Suppuration of both orbits as the result of a boil on the bridge of the nose. On the right side the abscess was subperiosteal and the ethmoid cells became secondary infected. On the left side the pus was confined to the cellular tissue of the orbit.*

G. D—, æt. 16 years. On Christmas Day, 1916, the patient developed a boil on the bridge of the nose, which suppurated and was opened several times by her doctor. A fortnight after the appearance of the boil the left eye

became slightly proptosed and then went down. At the same time the right eye became proptosed and both upper lids became red and painful. There was no history of any nasal trouble. She was admitted to the Central

FIG. 25.

Lacrymal abscess pointing above the tendo-oculi.

London Ophthalmic Hospital on January 13th, 1917. There was proptosis of both eyes, more on the right side than the left, and both lids were red and œdematous ; the eyes were pushed somewhat downwards, and there was marked limitation of movement in both. Temperature, 100·4° F. Pulse, 140. Respiration, 26. The nose was examined both by rhinoscopy and the X rays ; there was nothing abnormal to be seen. On January 13th an incision was made down to the bone of the right upper and inner

orbital margin. The periosteum was stripped up and an abscess opened and drained. The bone of the ethmoid was found to be bare and rough in one situation. The temperature did not go down. On January 16th, the left eye had become more proptosed and the right was still bad. The patient was seen in consultation with Dr. H. L. Lack. The left orbit was opened by an incision along the supraorbital margin down to the periosteum, which was stripped up over the whole of the inner surface of the orbit, but no abscess was found and the wound was closed by sutures. In the right orbit the wound was enlarged and the ethmoid explored. The ethmoid cells were opened up and found to be full of creamy pus. The anterior end of the middle turbinate was removed and a drainage-tube passed through from the wound and out at the anterior nares (Fig. 23). On the 18th there was a marked improvement in the right eye, but the left was worse; the proptosis greater and there was a small opacity in the cornea of the lower part. During the whole time both eyelids were stitched together to preserve the cornea as far as possible. On the 20th the right orbit was much better. The left eye had become more swollen, and under chloroform another search for pus was made; the old wound was opened up, the cellular tissues of the orbit explored with sinus forceps, and a large abscess was opened on the outer side of the orbit behind the lacrymal gland and a drainage-tube was inserted. On January 22nd the right eye was very much better and the proptosis was subsiding; the left eye was more proptosed and the upper lid more œdematous. Exploration with sinus forceps was again made in the cellular tissues, and a large abscess containing about two or three ounces of pus was opened on the inner side. A counter-opening was made in the lower orbital margin and a tube passed through from one to another. After opening this abscess the temperature, which had been varying between 100 and 102° F., subsided, and the proptosis gradually began to go down. On January 31st, a small superficial abscess was opened below

the left eyebrow near its inner end. On February 2nd, the right lower lid became swollen, red, and œdematous near its inner end. An incision was made and pus was found, which was evidently a secondary abscess from the necrosis of the orbital plate of the ethmoid. On February 6th another secondary abscess was opened in the lower lid of the left side. The pathological examination of the pus showed the inflammation to be of purely staphylococcal origin. On February 7th staphylococcus vaccine was given and subsequently repeated once a week. The tube passing through the nose in the right orbit was left out and subsequently a week later the tube in the left orbit was also removed, and the patient made an uninterrupted recovery. On examination on March 1st, neither fundus showed any signs of optic neuritis or other abnormalities. The vision of the right eye was $\frac{6}{6}$ and the left $\frac{6}{12}$. The latter vision was accounted for by a slight corneal nebula due to exposure and is rapidly clearing (Fig. 24).

Case 2.—*Case of frontal sinus suppuration simulating a lacrymal mucocele.*

E. C—, æt. 50 years, female, has been troubled with "growths" in the left side of the nose for years. She was admitted to the Central London Ophthalmic Hospital with a large left lacrymal mucocele, which, when you pressed over the sac, emptied itself into the nose.

On February 18th, 1908, the sac was excised, and the pathological report says that the mucous membrane shows signs of chronic inflammation, and the sac wall is much thickened but no sign of tubercular origin. Three months later the patient came back to the hospital with a history of having considerable pain over the eyebrow and a return of a similar swelling in the situation of the old mucocele. On pressure over the sac and making the patient bend her head forward pus came down the nose. It was thought that there was a relining of the cavity with epithelium and a fresh mucocele formed. She was again admitted to the hospital and an incision was made over the swelling. On opening the cavity it was found there was a track

passing upwards towards the frontal sinus into which a probe could be passed, and there was a hole in the lacrymal bone which passed into the nose. The swelling, therefore, was a collection of pus which had escaped from the hole in the frontal sinus, gravitating downward into the lacrymal region and then drained out through a hole in the lacrymal bone into the nose. The patient was subsequently transferred to a throat hospital for treatment.

Case 3.—*An abscess in the right eyebrow simulating frontal sinus suppuration.*

Male, æt. about 30 years. Had attended throat hospital with some nasal trouble. For two weeks before he was seen at the hospital he had had intense pain over the right eyebrow. On admission he had a large fluctuating swelling situated in the lower part of the eyebrow and upper part of the orbit. The eye was somewhat displaced downwards and the swelling seemed to pass backwards into the upper part of the orbit, but it was so large that digital examination was very difficult. The nose was examined, but showed no sign of any disease. An incision was made into the abscess and the pus evacuated, and the abscess cavity was found to be superficial to the periosteum. Recovery was uninterrupted (Fig. 22).

Case 4.—*Suppuration (abscess) in Tenon's capsule (2 cases).*

M. C—, female, æt. 34 years. Married. On January 10th, 1916, the patient was taken ill with sickness, pains in the back, and a temperature. On January 14th she developed an acutely painful, swollen right ankle-joint, the skin of which was red and inflamed. At the same time the left eye became painful and proptosed. On January 15th I first saw the patient with her doctor. The right ankle-joint was enormously swollen, painful, and inflamed. Her temperature was 101° F. The right eye was proptosed and pushed straight forward, and the conjunctiva œdematous; the iris was somewhat discoloured and beginning to show signs of inflammation. She could count fingers with the eye. On January 18th the proptosis had increased.

There was marked iritis and a purulent infusion into the vitreous which could be seen by focal illumination. Under an anæsthetic incisions were made into the upper and lower lid, and the orbital tissues explored with sinus forceps without any result. After the operation the proptosis increased, and on January 24th an abscess burst through the conjunctiva between the insertions and external and superior rectus. After this the proptosis gradually subsided and the iritis gradually settled down. The purulent exudation in the vitreous could be still seen. The ankle-joint had subsided without suppuration. Nine months later the eyeball showed some signs of shrinking; the anterior chamber was deep and the tension soft, but the lens was quite clear and a yellow mass could be seen behind it. The movements of the eye were fair in all directions.

Case 5.—*Inflammation of Tenon's capsule (pyæmic), secondary to cystitis due to Staphylococcus aureus. Pus in vitreous sterile.*

D. S—, male, æt. 65 years. Admitted to Central London Ophthalmic Hospital, January 11th, 1916. Married. Three children. All living well. Has been well all his life; never had any trouble with his eyes before. First noticed left eye misty two days before admission. Shortly after it became red and the lids became swollen. No pain. Present condition rather emaciated. Urine, specific gravity 1025 acid. Albumin and blood present (cystitis). Sugar *nil*. Left eye swollen and œdema of lids and conjunctiva. Conjunctiva overlapping cornea and limbus. Eye slightly proptosed. Some limitation of movement in each direction. Cornea clear, lens clear, fundus hazy, due to vitreous opacities; some iritis. Vision, P.L. Temperature, 98·1° F.

January 12th.—Œdema of the lids. Left eye more proptosed; exudate on the anterior surface of the lens and purulent matter could be seen in the vitreous. Temperature, 99° F.

January 18th.—Incision made in upper lid. Upper

part of orbit explored; nothing found. Drained, and lids stitched together.

January 21st.—Considerable increase in the swelling of the lids, and proptosis and exudate in the pupil and vitreous. Eye still has perception of light.

February 4th.—Enucleation of left eyeball; small abscess was found to the inner and lower side of the optic nerve. Temperature, 99° F.

February 8th.—Temperature, normal.

Cultures from orbit show *Staphylococcus aureus*. Pus from the eye sterile.

Case 6.—*Suppuration in a lacrymal sac presenting above the tarsal ligament.*

Girl, æt. 12 years, came to the hospital with an abscess pointing above the internal tarsal ligament. She previously had lacrymal obstruction and epiphora for some two years previously. An incision was made into the abscess, which was found to communicate with the lacrymal sac and was drained (Fig. 25).

VIII. DISEASES OF THE CORNEA.

1. *Case of Mooren's ulcer treated with a conjunctival flap.*

By F. A. C. Tyrrell.

Eugene de la V—, æt. 38 years, attended Ophthalmic out-patient department, of St. Mary's Hospital, February 9th, 1917, with large ulcer extending over upper third of left cornea. Ulcer was of crescentic shape with concave edge undermining epithelium. Floor of ulcer shallow and grey, staining in patches, slightly diminished sensation, no pain. A typical Mooren's ulcer in its early stage.

The edges of the ulcer were cauterised the same day. No improvement could be observed after a week; ulcer somewhat extending at horns of crescent. The patient was taken into hospital for further treatment and cautery again applied without success. After four days I decided to cover the ulcerated area with a conjunctival flap. The floor of the ulcer was first scraped with a sharp spoon and a flap cut which was stretched over ulcer and stitched with three sutures to uncut conjunctiva below. The central stitch resting on healthy cornea was removed within twenty four hours for fear of injury to epithelium. The flap failed to adhere completely in central part leaving a small area uncovered. Numerous blood vessels however soon bridged the gap. The rest of the cornea looked bright, healthy, and well nourished a week after the operation. The growing horns of the ulcer were well covered and ceased to extend, and the small area of staining gradually diminished. Patient was discharged after a few weeks with a lozenge shape nebula

just above papillary area covered with healthy epithelium. The upper limbus was highly vascular and prominent, but was under cover of eyelid to a great extent.

He was called up and passed into the French Army at the end of April, ten weeks after commencement of treatment.

Mr. Leslie Paton: This case when I saw it first was a typical Mooren's ulcer involving a crescent which extended round one third of the corneal circumference in the upper edge. It showed rather a shelving edge peripherally with an abrupt almost overhanging edge internally.

Corneal sensibility was only slightly diminished and there were no greyish infiltrates in the cornea beyond the crescent. Two or three areas in the crescent stained with fluorescin. The condition was comparatively painless. The whole crescentic area was cauterised twice at an interval of a week without improvement. Mr. Tyrrell then covered the area by bringing down a large conjunctival flap from above. This adhered at the outer and inner edges and from these two points the small remaining oval area speedily became vascularised and at the end of a month he had a quite smooth clear cornea. Mr. Tyrrell has since done a second case of very advanced Mooren's ulcer in which only a small island of clear cornea was left in the centre about 5 mm. + 4 mm. It is as yet too early to speak about the final result in this case, but where the flaps have adhered there is already a marked improvement in the condition. I think our experience with these two cases is sufficiently favourable to suggest to other members the desirability of giving a trial to this mode of dealing with these heart-breaking cases.

The President said that they would welcome any treatment which offered any prospect of success in dealing with cases of Mooren's ulcer. He had heard previously of this case of Mr. Tyrrell's, and as he had a bad case of Mooren's ulcer in the hospital, he asked Mr. Tuckett, the

house surgeon, to deal with it in the way recommended. The patient, however, got dissatisfied with being in the hospital and discharged himself, so that he lost sight of the case and the ultimate result of the treatment was not known. The case was a very advanced one, and he thought the treatment seemed likely to be beneficial.

Mr. ROWAN asked whether there was any difficulty in trying the use of the central stitch instead of a cross suture. He had tried this often in injured eyes. The suture was just passed in and out and the two ends tied together. The stitch in this case did not seem to touch the cornea. It is often called a "Purse" suture.

M. J. G. CLEGG said that when the ulcer was covered up with the conjunctival flap, the tension especially on the sides—there was a less amount of tissue laterally—sometimes caused the purse skin thread to cut through the conjunctival substance. In a case of his own, a day or two after the operation had been done—at all events by the third day—the conjunctiva had slipped back.

IX. DISEASES OF THE UVEAL TRACT.

1. *A case of "uveo-parotitis" with irido-cycloplegia.*

By George Mackay.

On March 10th, 1916, Maggie R—, æt. 30 years, a tablemaid, was brought to me by her mistress. She looked well-nourished, and there was nothing exceptional to note in her general appearance except that she was slightly anæmic, depressed, and anxious about the condition of her eyesight. She had been in the same situation for eight or nine years, and was a well-conducted and highly-valued servant.

History.—At the age of 7 or 8 years she had measles, and since then had been liable to slight blepharitis marginalis and on that account had sometimes to be kept from school in winter if the weather were cold. At the age of 11 years, being a native of Montrose, she went into a factory for the making of yarn from flax, and worked there for ten years. Thinking that this rather dusty occupation was keeping up the lid weakness, she decided to leave the factory and engage in domestic service. She was in the habit of using boracic lotion for her eyes and occasionally Singleton's ointment. In other respects she had no complaint about her eyes or eyesight until the onset of her present illness. She had never had mumps, and, so far as she knew, prior to her present illness, had not been in contact with any persons suffering from mumps. At the age of 20 years she had all her teeth removed. She had enjoyed good health until a fortnight before coming to me, when she had what she described as a "cold" with cough. There had been no

sore throat nor any known contact with diphtheria. On further inquiry it was admitted that there was not much catarrh in the head, but rather a feeling of malaise and pains about the stomach, with a certain degree of nausea, so that she feared to eat lest she should be sick. A day or two afterwards there was some swelling of her eyelids and soon thereafter a difficulty in reading small type in the newspaper. This became aggravated, so that a few days later she could not see even the larger headings of the paragraphs. There was an inclination to close the eyes, apparently due to slight photophobia.

Present state.—When I first saw her on March 10th, 1916, there was a slight diffuse, deep conjunctival, and very moderate ciliary injection. The pupils were rather widely dilated (about 6 mm. in diameter) and immobile to light as well as on convergence of the visual axes, and though appearing almost circular, on closer inspection a few posterior synechiæ were found. These had apparently formed while the pupils were in a state of dilatation. They were sufficiently eccentric in position to suggest that a mydriatic had been applied previous to their formation. This seemed all the more probable since, on further examination, it was found that her accommodation was apparently paralysed. There was well-marked punctate keratitis on the back of both corneæ, and haziness of the vitreous. Through this fog the details of the fundus were rendered somewhat obscure, but no gross changes were to be seen at the disc or elsewhere.

R.V. = $\frac{5}{60}$, not improved by any lens.

L.V. = $\frac{6}{60}$,, ,,

At 10 in. her vision was relatively worse. The right eye could barely decipher J. 19, the left J. 16. With + 4 D. sph. improvement was got to about J. 14 and J. 12 respectively. There was undoubtedly mydriasis, and there appeared to me to be certainly cycloplegia as well, though the turbid media made the precise degree difficult to determine. Despite most careful inquiry I could not elicit that she had been using any mydriatic drops or

ointment, nor handling any plaster or liniment or medicine which might have contained belladonna or any such cycloplegic. Her tongue was fairly clean, her appetite poor. The temperature did not appear to be raised. This association of iridocyclitis with paralysis of the pupil and accommodation appeared to me so exceptional that I asked my friend, Dr. F. D. Boyd, the family physician, to see the patient. On March 13th she was admitted under his care to Ward 24 of the Edinburgh Royal Infirmary. I requested that precautions should be taken to see that there was no possibility of her having access to any cycloplegic and that every possible means should be employed to ascertain by bacteriological examination whether the disease had a microbic origin. Two or three days after admission she developed bilateral swelling of the preauricular portions of her parotid glands. The cervical part of the parotids were less affected and no other glands in the neck were involved. The enlarged parotids were smooth, firm, and only slightly painful on pressure or on yawning. Her temperature, which was 98·2° F. at admission and 99° F. the following morning, remained for the most part rather subnormal, varying from 96·8° F. on several occasions in the morning to generally a degree more in the evening. The highest temperature recorded was 99·4° F. on the 19th—two days after having had a von Pirquet's cuti-reaction applied. On March 29th, Dr. Fitzgerald, one of the pathologists to the Royal Infirmary, reported:

(1) That she was negative to Wassermann's test.

(2) That she was negative also to von Pirquet's cuti-reaction, and that a subsequent tuberculin injection of Koch's old tuberculin had also given a negative result.

(3) That no growth had been obtained from the blood culture.

(4) That the blood showed a leucocytosis.

(5) That a culture from the conjunctiva showed Gram positive cocci (*Staphylococcus albus* and *citreus*).

The hospital notes do not definitely state whether any

swab was taken from her throat. If it was done, it was certainly found negative. Unfortunately, the period of her admission coincided with a change in the period of office of resident physicians, and, owing partly to the pressure of war-time demands, there was some divided responsibility in the recording or neglect to carry out this observation. I could elicit no history nor symptoms pointing to any ulcerated condition of the throat, and I can only add that having myself taken a swab from her throat on May 12th, it was reported by the Laboratory of the Royal College of Physicians to contain no diphtheria bacilli. This is perhaps a rather belated observation. At the same time, knowing that long after an acute attack of diphtheria has subsided bacilli may still be found in the throat, I give it for what it is worth. By April 7th the patient said that her eyes were improving. She could tolerate light with less discomfort, and the parotid swellings, though still obvious, had considerably diminished in size. On the following day, April 8th, the patient was dismissed from hospital, *i. e.* nearly a month after her first visit to me.

On April 24th (*i. e.* six weeks after her first visit) I saw her again at my house. The pupils were now rather more oval, with longer axes horizontal. The parotid glands still felt slightly indurated on palpation between the jaw and ear, though there was now no obvious swelling. I think I may safely assert that they were not normal for fully a month and a half in her case. There was apparently some deficiency in the salivary secretion at this time, for she complained that she had difficulty in masticating and swallowing dry food unless she took some fluid with it; but the act of deglutition was not impaired and her speech was not nasal. There was much deep punctate keratitis, the spots having then that tawny colour which usually indicates recession of the exudate. The vision for test type was just as previously recorded.

About May 12th (9th week), the vision was at its worst. The right fundus could no longer be seen.

R.V. = fingers at 3–4 metres.

L.V. = $\frac{6}{36}$, with difficulty.

Thereafter improvement set in. On May 30th she expressed the opinion that her vision for objects on the street and for the furniture in my room was returning, but the actual response to test types was practically unchanged. There was still much punctate keratitis with floating opacities in the vitreous. I did not see her in the course of the summer, though I heard that she was getting better, and I had no further opportunity of examining her until November 9th, when I found:

V. = $\frac{6}{36}$. Improved with + 0·5 D. sph. to $\frac{6}{24}$ partly, in each eye.

With an effort J. 6 was read at 10 in. unaided by glasses and there was a visible narrowing of the pupils during this act of accommodation. The relative improvement in near vision was remarkable, and strongly supports the view that there had been profound cycloplegia earlier. When at rest, facing the light and looking directly forward, the pupils were circular 5 mm. in diameter, *i.e.* a little smaller than formerly. There was no evident contraction even on exposure to focussed light, so that the ciliary muscles, aided by voluntary impulse, appeared to be regaining their action somewhat in advance of the reflex activity of the sphincters of the pupils to the stimulus of light. There was still considerable brown punctate keratitis and pigment spots on the anterior capsules of the lenses, with floating opacities in each vitreous, but the optic discs could now again be seen, the right rather more easily than the left. There was no evident choroiditis.

On February 1st, 1917, the whole condition had further improved and

R.V. = $\frac{6}{18}$.

L.V. = $\frac{6}{12}$ partly; while with each eye unaided she could now decipher J. 1.

My last examination was on April 19th, when I found that with the right eye she could read $\frac{6}{12}$ (save one letter) and J. 1. With the left eye, $\frac{6}{12}$ correctly and J. 1

more easily. The pupils were equal and almost if not quite circular, especially the left. Each appeared about 5 mm. in width in subdued light and responded very little if at all even to brightly focussed light, but definitely narrowed to about 3 mm. on convergence with accommodation. She still has some fine brown spots of deep punctate keratitis on both corneæ, and on the lower part of the right cornea one larger grey greasy spot composed of a small chain of more recent confluent deposits forming an incomplete ring about 2 mm. in diameter, shaped like a C or one of Landolt's vision test figures. The vitreous is now practically free from opacity in both eyes; the optic discs are well defined and there are no visible choroidal changes to be seen even in the periphery of the fundus. Nothing abnormal is to be felt about the parotid glands; salivation appears to be quite natural; the ciliary muscles have practically regained their power. On the whole, she seems in a fair way to make a complete recovery, but it is now more than a year since the onset of the disease. No mydriatic has been employed in treatment—only dark glasses and tonics.

Commentary.—I must confess that when this case first came under my observation prior to the development of the parotitis, I was at a loss to explain the concomitance of irido-cyclitis and irido-cycloplegia. The subsequent development of bilateral parotid swellings at once suggested that the case was one of mumps, but even if so it is unusual for eye symptoms to *precede* the parotid trouble. They usually follow as later symptoms. Perhaps I should say here that the case was quite unlike either Parinaud's or Mickulicz's diseases.

Ocular complications of parotitis epidemica have been frequently recorded in medical and ophthalmic literature. Thus: swelling of the lids; swelling of the lacrymal glands (for which Hirschberg has suggested the term "lacrymal mumps"); lacrymation; conjunctivitis; some rather dubious cases of keratitis; several cases of irido-cyclitis with keratitis punctata profunda generally with

vitreous opacities ; optic neuritis ; optic atrophy ; paralysis of extra-ocular muscles with associated diplopia and some degree of exophthalmus ; facial palsy involving the eyelids ; and more rarely mydriasis and paralysis of accomodation. From most of these complications the recovery has been satisfactory after a variable length of time, but optic neuritis, just like the more familiar metastatic orchitis or oöphoritis, may go on to optic atrophy and total loss of vision. In one case at least (Schiess-Gemuseus) an irido-cyclitis took so unfavourable a course that enucleation was performed to lessen risk to the other eye, and this supplies the only pathological report which I have discovered. It is not very enlightening, but so brief that I can give it you in eight words : " Iris extraordinarily thickened, peculiar pulpy tissue, much vascularisation." In addition to the ocular complications following mumps which I have above mentioned, there are, of course, many other grave complications. Paralysis of various parts of the central nervous system, involving both motor and sensory functions, have been recorded. I am not at present concerned to discuss these, and only refer to them as an indication of the fact that, though in the great majority of cases mumps is regarded as a harmless affection, it may be accompanied by complications which are temporarily, at any rate, of a serious nature. But again the question arises whether my case was truly one of mumps at all.

In that connection I should like to draw attention to a paper published in 1909 by Heerfordt in v. Graefe's *Archiv für Ophthalmologie*, vol. lxx, p. 254, entitled " On a Subchronic Uveo-parotid Fever, localised in the Parotid Gland and the Uvea of the Eye and specially complicated with Paresis of Cerebro-spinal Nerves." Heerfordt records three separate cases which occurred under the care of three different physicians in the Copenhagen Town Hospital in the years 1905–6. The first, a boy of eleven ; the second, a boy of fourteen ; and the third, a manservant of twenty-seven.

They were all cases in which, *without any known history of exposure to mumps*, but after experiencing for some weeks feelings of tiredness, drowsiness, loss of appetite, and so forth, parotid swellings developed, associated with iridocyclitis and various pareses of nerves. In the first and youngest of the trio, there were symptoms of pyrexia sufficiently pronounced to justify the employment of the term "fever," to which Heerfordt gives prominence in his title. During his stay in hospital for four or five months this boy's temperature varied from three to four degrees above normal. In each of the other two patients there was less fever—only a very slight, but protracted tendency to pyrexia.

The circumstances which seemed to justify the grouping together of these three cases were that whereas in mumps (1) the premonitory stage is usually brief (twelve to thirty-six hours), in Heerfordt's cases it varied from a fortnight to many weeks. (2) The parotid swelling, which usually subsides in two to eight days and may exceptionally continue for two, three, or even four weeks, was noted in his cases for more than two months. (3) The increased temperature which commonly varies with the severity of the attack is not usually of long duration in mumps and often falls suddenly by "crisis." In his cases it was not high, but more protracted. (4) In the first case, iridocyclitis and optic neuritis *preceded* the parotid swelling by about a week. In the other two, the ocular and other nerve symptoms *followed after* parotitis. Thus, in the second boy, there was iridocyclitis first in the right eye and later in the left, a transient right facial palsy, and for three weeks after admission difficulty in swallowing food.

The third case (man, æt. 27 years) complained of feeling out of condition and tired for some months before his admission to hospital. The parotid glands then became swollen, double irido-cyclitis developed, and other nerve symptoms followed, difficulty in swallowing, right facial palsy, and disturbances of sensation in the skin of the abdomen and hands, and diuresis. There is no record of

cycloplegia in any of this trio. Nor is there any record of diphtheria in either of the boys, but the report on the man concludes with the remark that diphtheria bacilli were found in the fauces. On this point Heerfordt makes no further comment, but came to the conclusion that if his cases were due to mumps they were very atypical, and he raised a question whether they ought not to be assigned to some hitherto unrecognised category, constituting a separate clinical entity.

In his survey of the literature Heerfordt fixes on two cases which he considers should be grouped with his own. The first concerns a military musician, æt. 22 years, seen in 1897 by Daiřeaux (2) and Pechin (3) eleven days *after* a bilateral parotitis—diagnosed as mumps, though there was apparently no epidemic of that disease at the time. He developed right facial palsy and loss of taste on the right side. This cleared up in about a month, but a week or two later a double iritis with some keratitis followed. The other case published by Collomb (4) appears to me to be of greater interest, since ocular trouble *preceded* parotitis. An apparently healthy man, æt. 29 years, after some digestive disturbance developed redness of his right eye. When first seen by Collomb a few days later he had parotid swelling and iridocyclitis, combined with hyperæmia of the disc. This was followed by a slighter attack in the left eye. In this case, so far as known, there was no exposure to epidemic parotitis.

In 1916, Kuhlefelt (5) reported "a case of subchronic uveo-parotid inflammation" in a Finnish journal published in the Swedish language at Helsingfors.* This is a case of a clerk, æt. 21 years, who consulted him on August 20th, 1915. Her father died of tuberculosis. She had mumps as a child. In adolescence some affection of the fourth metacarpo-phalangeal joint of the right hand. In July had some swelling and tenderness in each parotid region, followed by diminution of acuteness in the right eye. In

* I am indebted to Dr. Kuhlefelt for kindly sending me a copy of his paper, and to Sir George Berry for a translation of its contents.

the middle of August complained of dimness of vision in the right eye and injected conjunctiva, "Descemetitis," hyperæmia of the iris, media otherwise clear, but apparently neuro-retinitis with some turgid vessels, circum-papillary greyness, and eccentric retinal hæmorrhages. The vision $= \frac{5}{10}$. She evidently had irido-cyclitis with neuro-retinitis in the right eye, but no cycloplegia. Occasionally, slight rise of temperature. She had apparently not been exposed to epidemic parotitis. The left eye was normal.

In the *Lancet* of January 6th, 1917, p. 28, there is an editorial note on a paper by Leeksma, of Heukelum, published at Amsterdam last year, with reference to a married woman, æt. 28 years, who, after feelings of tiredness, some shivering, and night-sweats, developed pain in the left ear and then right facial paralysis. A week later the left parotid gland became swollen. A fortnight later the facial palsy cleared up, but was followed by herpes of the left cornea and that again by iridocyclitis. There was apparently no cycloplegia. On inquiry, Leeksma found that there were three typical cases of mumps in this patient's environment during the middle period of her illness (January to March), and he came to the conclusion that while his case resembled Heerfordt's, uveo-parotid fever is nothing more than atypical mumps and not a separate morbid entity.

I have myself made a fairly careful analysis of the papers referred to by Heerfordt in his communication of 1909, and have searched through the *Index Medicus* from that date up to the present time, and have only found details of six cases of dilated pupils and paralysed accommodation recorded in association with parotitis. In three of these the irido-cycloplegia appeared to be a secondary consequence of optic neuritis or optic atrophy, or as part of a third nerve paralysis, namely, in cases published by Talon (6) in 1883, Swan Burnett (7) in 1886, and Blanchard (8) in 1899. Of the remaining three cases the first, published by Baas (9) in 1886, was a little girl, æt.

7 years, who had cycloplegia three weeks *after* mumps. Diphtheria was excluded.

The second, reported by Mandonnet (10) in 1903, was a child, æt. 9 years, with complete paralysis of accommodation and of the soft palate *after* mumps. The third, recorded by Dopter (11) in 1904, was a guardsman, æt. 31 years, who, two or three weeks *after* mumps, which quickly subsided, developed pyrexia, occipital headache, and double orchitis, left facial palsy, paresis of left half of tongue and soft palate, left mydriasis and left cycloplegia. He had definite leucocytosis and made a good recovery. In none of these was there iridocyclitis.

I have not found any record of a case exactly reproducing either the group of symptoms or the order of their occurrence, which characterised the one under my own observation.

One must not overlook the fact that inflammation of the parotid gland is not a very uncommon complication of various septic medical and surgical conditions, *e. g.* typhoid fever, appendicitis, and abdominal operations. This gland is a channel for the excretion of deleterious material. Various drugs make their escape by the saliva, and an abundant salivation is a common precursor and accompaniment of bilious vomiting. The ciliary body doubtless plays its part also in endeavouring to get rid of deleterious substances circulating in the blood-stream. It appears to me not improbable that the obstinate and tedious character which iridocyclitis so often presents is due to the fact that the morbid material passing from the ciliary gland into the aqueous fluid has no chance of being directly evacuated from the body like the saliva, but is necessarily reabsorbed into the circulation and sets out again in a vicious circle. It is commonly believed that in mumps the poison enters the parotid gland through the buccal orifice. If that be so, it is easy to understand how the parotid gland reaction precedes other signs and symptoms of remoter or metastatic inflammation or nerve pareses which are presumably due to an extension of the poison

through the system or are set up by toxins produced by the primary infecting agent. When remoter toxic effects *precede* the parotitis I venture to suggest that the poison, whether due to mumps or not, may (or must) have entered by some other portal than Stenson's duct. The possibility of a mixed infection, *e. g.* with diphtheria, should not be put aside without further careful investigation.

Bearing all these problems in mind there is undoubtedly some justification for Heerfordt's scepticism as to whether there may not be another ætiological factor than mumps for cases such as he and I have recorded in which no association with mumps has been established. This problem cannot be correctly solved until pathological investigation has determined what is the precise cause of mumps, as certainly as it has revealed the cause of anthrax or tetanus or diphtheria. Personally, I shall suspend judgment, and, adopting the cautious procedure of our Scottish Courts, go no further than to say that the case for mumps as the sole cause for uveo-parotitis is, as yet, "not proven."

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(3) Pechin.—"Complications oculaires des oreillons," *Recueil d'ophthalmologie*, Juni, 1901.

(4) Collomb.—"Un cas d'iritis ourlienne," *Revue medicale de la Suisse romande*, June 20th, 1903.

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companying Mumps," *International Journal of the Medical Sciences*, 1886, vol. xci, p. 86. (This is now the *Amer. Journ. of Med. Sci.*)

(8) Blanchard.—*Bulletin Medical*, 1899, p. 1091.

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(10) Mandonnet.—" Paralysie de l'accommodation et du Voile du palais, consécutive aux oreillons," *Ann. d'ocul.*, 1903.

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2. *Iridocyclitis of obscure origin.*

By D. Leighton Davies.

In this short paper I propose to deal with a condition resulting from chronic infective processes of an unusual type; I refer to the results of infection of the anterior part of the uveal tract, *i.e.* the iris and ciliary body.

The ætiology of iridocyclitis is apt to be one of the most obscure problems that we meet with in the course of our ordinary daily work. Some forms of iritis have an easily ascertainable origin; others may baffle the most careful and painstaking investigation, but, thanks to the patient labours of many investigators, our knowledge of the primary source of infection in diseases of the iris and ciliary body is greatly in advance of what it was, say, twenty years ago.

There must be few ophthalmic surgeons to-day who are satisfied having diagnosed a case as "rheumatic iritis." It is a nice comforting diagnosis from the patient's point of view, because he or she thinks that explains everything and it is a popular complaint. But although the term "rheumatic iritis" is a useful one, it should not blind us to the fact that we have not arrived at the ultimate cause of the condition when we use this term. Again, the term "gouty iritis" is a very loose one and conjures up but a very vague mental picture of the pathogenesis of the

iritis. Probably true gouty iritis may have existed at a time when acute gout was a prevalent disease amongst the middle-aged and elderly, but acute gout is, I believe, very uncommon nowadays, so uncommon indeed as to be almost a rarity.

Acute inflammatory diseases of the uveal tract are, apart from injuries, almost always of infective origin, but it is not always so easy to prove this statement as to make it. The infection may be part of some other general infective disease *i.e.* syphilis, tuberculosis, typhoid fever, and so on, or it may be the result of an auto-intoxication arising from various regions of the body.

These may be :

(1) *The alimentary system.*—In the mouth pyorrhœa alveolaris is recognised as a fertile source of infective processes. The tonsils again, when the seat of chronic infection, may be the cause of an iritis, as has, I believe, been shown. Lower down the tract intestinal fermentation has been shown to be a cause of iridocyclitis. This was ably demonstrated by Mr. Lawford and others in the debate on alimentary toxæmia at the Royal Society of Medicine in 1913.

(2) *The respiratory passages* : (A) The accessory nasal sinuses. I have seen a case where a constantly relapsing iritis was cured by operative treatment for pansinusitis which had its origin in a frontal sinus suppuration. The patient had refused to undergo the radical cure of her nasal suppuration for many years, during which time she had frequently recurring attacks of iridocyclitis which brought her sight down to less than $\frac{6}{60}$ in each eye. Since operation, now two years ago, she has had no relapses, although, owing to vitreous opacities and old deposits on the lens, capsule vision does not improve. (B) A chronic empyema or bronchietasis may equally well produce the condition, though I have not come across such a case.

(3) *The genito-urinary tract.*—Under this head gonorrhœa may be counted the most important factor. In the

male a long-forgotten attack of gonorrhœa may be the cause of an obstinately recurring iritis. I have notes of a man who has had slight attacks of iritis (about twice a year) for many years. He owned to a slight attack of gonorrhœa twenty years previously, and after massage of the prostate, prostatic plugs and gonococci were found in the urine. Vaccine treatment decreased the frequency of the attacks, but did not cure him. In women proof is not so easily obtained, but undoubtedly is a factor that must ever be borne in mind. These cases are not at all easy to treat. Some seem to respond very well to vaccines, others do not respond at all. Probably reinfection takes place from the prostatic crypts, from whence it is almost impossible to expel the gonococci.

My object is, however, to draw your attention to the bladder as a possible cause of an attack of iritis, when no obvious cause of infection is to be found. Let me quote very briefly the notes of three cases.

(1) C. A. L—, spinster, æt. 45 years, consulted me in September, 1914. She gave a history that a fortnight previously one day she found she could only see one half of things at times. At first this phenomenon lasted only a few minutes at a time, but later on in the day it persisted almost without intermission. The following day the left eye was misty, and two days later it became bloodshot and painful. She had had a transient attack of "one-sided blindness" followed by severe headache when fifteen years old, and had had two other attacks of inflammation in the right eye previous to the present attack. There was a history of slight constipation. Examination: V., R. eye $\frac{6}{12}$, with $-0{\cdot}25$ D. sph. $\frown$ 0·75 D. cyl. $\downarrow$ 120° $\frac{6}{6}$; L. eye $\frac{6}{24}$, J. 2 in each eye. In the left eye there was no injection, but numerous fine spots on the lens capsule in the area of the pupil, and some vitreous opacities. The iris appeared to be normal. She was wearing an artificial denture, underneath which were several stumps. She also had a naso-pharyngeal discharge, and much leucorrhœa; the urine was normal. She looked ill, and had been

losing weight for some time, and had suffered pains in the back and shoulder, etc. All the stumps were removed from the mouth as soon as possible. The nasal sinuses were found to be clear and the naso-pharyngeal discharge soon improved under simple nasal douching. The usual local treatment was ordered for the eye, and she was told to take small doses of Epsom salts daily. In spite of this the left eye got worse, and at the end of a month the right eye showed marked signs of iridocyclitis, with numerous "mutton fat" deposits on each cornea. In March, 1915, *i.e.* six months later, she was still doing badly and losing weight. At that time some crepitations were found in the right lung, but no tubercle bacilli were found in the sputum. In spite of this it was thought wise to try tuberculin injections. This was accordingly done, but with no effect on her general health or the course of the iritis. In May she was still losing weight rapidly ($2\frac{1}{2}$ lb. in 14 days) and vision had sunk to — R. eye $\frac{6}{60}$, L. eye $\frac{6}{18}$. No details could be obtained of either fundi on account of the vitreous haze. A further careful general examination at the end of June resulted in the isolation of a coli-like bacillus from the urine, and she was advised to have vaccine treatment. This was started in July, 1915, the injections being given at intervals of a week to ten days. After the sixth injection her sight began to improve. By September, 1915, it was: R. $\frac{6}{12}$, L. $\frac{6}{18}$. There was still K.P. and vitreous opacities in both eyes, but the fundus could be seen in the right eye and appeared normal. The last injection was given at the end of October when her sight had further improved in the left eye to $\frac{6}{12}$ (1). Thereafter her general health improved and she began to put on weight, but she continued to have transient attacks of pain and mistiness in one or the other eye, kept in control, however, by atropine. On the other hand sight continued to improve steadily up to July, 1916, when she had vision R. eye $\frac{6}{6}$ (1) L. eye $\frac{6}{6}$ (1). There were then no K.P. to be seen in either eye and just a few very fine opacities in the vitreous; fundi both normal.

(2) The second case was that of E. E—, spinster, æt. 25 years, working as a jobbing tailoress. When seen in August, 1915, she complained of mistiness of sight which had lasted six months, she also had suffered from epilepsy for nine years. Her vision was: R. $\frac{6}{12}$, J. 2: L. $\frac{6}{18}$ (1), J. 2. She had a slight amount of mixed astigmatism, correction of which improved the left eye to $\frac{6}{12}$.

On examination under a mydriatic the pupils dilated well, but there was a large adhesion of the left iris, and in both eyes deposits on the lens capsule together with some fine K.P. The fundi were normal. Her vision gradually got worse, but no cause for the iridocyclitis could be found until November, when the urine was examined bacteriologically and found to yield pure cultures of the bacillus coli. At that time her vision was: R. $\frac{6}{36}$, L. $\frac{6}{60}$. There was then marked K.P. and vitreous opacities in each eye. Autogenous vaccines were given, but, except that her health improved, she never responded to them. I then lost sight of her for some months, but saw her again last March. Her condition was much the same, but her vision had deteriorated R. $\frac{6}{60}$, L. $\frac{4}{60}$. At no time have I ever seen any redness of the eye.

(3) J. G. J—, spinster, æt. 18 years. I first saw her in September, 1915. She gave a history of constantly recurring headaches for twelve months. Ten days before consulting me her eyes became inflamed, the left first and then the right, at the same time her sight began to get blurred. Her general health had been good.

Examination.—Vision: R. $\frac{6}{12}$, J. 2; L. $\frac{6}{12}$ (1), J. 2. No refractive error was present. The R. eye showed marked K.P., with a normal and active pupil. The L. eye showed slight ciliary haze, a large amount of K.P.; so thick, indeed, was the lymph that it was hanging down in the anterior chamber from the back of the cornea and there was a little hypopion. The pupil was sluggish, and new-formed blood-vessels were present on the surface of the iris.

In both eyes some vitreous opacities were present. No

cause of sepsis was found until the *Bacillus coli* was isolated in pure cultures from the urine. She was at once given autogenous vaccine injections. In three weeks' time there was no injection of the eyes, the K.P. had practically disappeared, and the vitreous seemed clear. I last saw her in November, 1915, *i. e.* two months after her first visit, and at that time her vision was: R. $\frac{6}{12}$, L. $\frac{6}{9}$ (1). I understand that she has remained well since. Of course, in addition to the vaccines, she had atropine for the eyes.

I have had three or four other cases in which the *Bacillus coli* has been found in pure cultures from the urine, but I have not included those cases, as the investigation was not completed nor had vaccine treatment been carried out. There are just a few points with regard to the cases I have seen and which I should like to emphasise.

Most of these cases occurred in women, and they have not uncommonly been associated with some constipation, though in the second and third cases I quoted this was not a prominent feature. But in view of the presence of constipation there is a flaw in the proof, for the infection may come from the alimentary tract primarily and not through the bladder, because, of course, the intestinal mucous membrane is more likely to be a good absorption area than that of the bladder. In view of that fact I always prescribed daily doses of Epsom salts, but without effect upon the course of the disease until the vaccine treatment was commenced. The last case I quoted, however, is striking in the rapidity of the cure. This, however, might have been so even if no vaccine treatment had been given.

The cases are not numerous enough to prove anything, but they are suggestive, and I have brought them forward because it may possibly lead to other observations on the influence of unrecognised bladder infections as a cause of chronic inflammatory infections of the uveal tract. The colleague with whom I was working and who was doing the bacteriological work has been called away on military

duties, so that the work is for the present suspended, but I hope in the day of peace to come to resume this line of investigation.

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3. *Two cases of herpes zoster affecting the ciliary nerves.*

By M. S. Mayou.

(With Plate IV, figs. 2 and 3.)

Case 1.—Miss P. B—. Commencing October, 1916. The patient had a severe attack of herpes zoster affecting the first division of the fifth nerve, especially along its supra-orbital and supra-trochear branches on the right side. The nasal branch was not affected. The attack followed influenza. On March 27th, 1917, she developed an acute iridocyclitis. The cornea in its deeper layers was steamy, and there was wrinkling of Descemet's membrane. The iris was discoloured, and there was a bright uniform ciliary injection with a slightly minus tension and no indication of the situation of the vesicle. Treatment was used of atropine and hot bathing, and the condition improved comparatively rapidly. Two weeks after being first seen the drawing was made (Pl. IV, fig. 2). At this time the steamy condition of the cornea had nearly entirely cleared up. There was some keratitis punctata and considerable exudation into the vitreous. Down and outwards in the sclera there was a raised nodule of a deepish violet colour, with some injection of the vessels around it. The remainder of the sclera had now become practically normal. This nodule was evidently the situation of an herpetic vesicle which occurred on one of the anterior ciliary nerves.

Case 2.—Mrs. D—. In January, 1917, the patient suffered from a severe attack of influenza, which was followed by an attack of herpes zoster of the first division of the fifth nerve on the left side, which affected the supra-orbital and supra-trochear nerves together with the

nasal branch. At the beginning of March she was seized with a severe pain in the left eye, and when seen by me on March 9th there was an acute ciliary injection with a hazy area in the cornea below, but the opacity was not entirely limited to the deep surface, but was also superficial and the epithelium over the surface was roughened; there was little or no iritis present, the pupil being evenly dilated with atropine. It was treated with atropine and hot bathing, and the eye tied up. On March 23rd a drawing was made (Pl. IV, fig. 3). The ciliary injection had largely subsided to the area affected. The cornea in its lower part was still opaque, but had become much more limited in its extent. In the sclera opposite the opacity in the cornea was a small nodule, which was raised and covered with dilated vessels.

Remarks.

The points of special interest about the above cases are that they both occurred in patients who had previously had influenza, and it is supposed by some observers that the toxin of the influenza bacillus is the cause of the eruption along the nerves (A. G. Shera, *Lancet*, March 24th, 1917). The other point is the long interval in which the eye becomes affected after the onset of the attack of the herpes.

4. (?) *Colloid nodules in the choroid in a girl.*

By M. S. Mayou.

(With Plate I, fig. 2.)

A. W—, female, æt. 25 years, single. She has worked in T.N.T. factory for fifteen months. During the last six months the sight of the left eye has been failing. She has also suffered from severe headaches and toothache during this time. The urine shows no sugar or albumin, and the heart, lungs, abdomen, and nervous system are normal. There is no family history of defective vision.

Vision in March, 1917: R. eye, $\frac{6}{9}$ + ·5 cyl. ax. vertical, $\frac{6}{6}$; L. eye, + ·5 cyl. ax. vertical, $\frac{6}{18}$. The lesion in the fundus is present in both eyes, but is rather more marked in the left, and there is a stippled condition of the macula in that eye not present in the other. The lesion consists of roundish white areas situated principally along the course of the vessels and distributed some considerable distance around the macula region (Pl. I, fig. 2). There is practically no pigmentation around them, although in one or two places there is a slight disturbance of pigment. There is no definite change in the visual fields.

The clinical appearance somewhat resembles the colloid degeneration of Bruch's membrane, which is seen in old people. A similar case was shown by Mr. L. V. Cargill before this Society (*Trans. Ophth. Soc.*, xxii, p. 162).

Mr. Mayou said that he had put a query before this case, though he did not think the case was very doubtful. There were greyish-yellow nodules scattered principally along the vessels. A point of interest about this patient was that she was a worker in T.N.T., but he did not think it probable that this had anything to do with the condition. The condition was present in both eyes, but there was failure of vision only in the left eye, and here the macula had a stippled appearance which the other eye did not show. He would be glad of any suggestions as to diagnosis and treatment. In reply to a question, he said that there was no history of family inheritance in this case. The blood showed no changes.

Mr. Grimsdale said that he thought this case was the early stages of the condition known as Doyne's Family Choroiditis, *Trans. Ophth. Soc.*, vol. xxx, p. 274. He had under his care at the present time a family suffering from the disease exhibiting a somewhat similar clinical appearance.

The President said that he had had an opportunity of examining one of Mr. Doyne's cases microscopically, and found the condition due to large hyaline nodules on the

inner surface of the elastic lamina of the choroid. The white patches in the fundus were much bigger than in the present case.

Mr. WHITEHEAD asked whether a blood-count was taken; they might be due to some changes in the constitution of the blood.

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5. *Leuco-sarcoma of choroid and epithelioma of lip, in the same patient.*

By J. A. VALENTINE.

E. B—, æt. 55 years. Epithelioma of lip removed with glands of neck, on the right side, in October, 1915, at the Portsmouth Royal Hospital.

More than a year later, in October, 1916, she came to the Eye and Ear Hospital, Portsmouth, complaining of loss of vision *in the left eye.*

The right eye had vision: Fingers at 1 metre. The lens was cataractous; the fundus showed a myopic crescent.

The left eye had vision $\frac{6}{36}$. Media clear, conjunctiva, cornea, anterior chamber, and iris appeared normal. There was a large limitation of field on the temporal side, when tested roughly by the hand, and she complained of seeing "lights" to the same side. On examining the fundus, a rounded tumour was seen, towards the nasal side, which almost reached the margin of the disc. It appeared to be a sarcoma, but was lighter in colour than the usual appearance of choroidal sarcoma. Small vessels could be seen bending round the curve of the tumour.

Measurements of growth:

Vertical height of section . 10 mm.
Antero-posterior diameter . 9 mm.

Colour white, like a section of nerve.

The tumour eye was excised on November 14th, 1916, and a section made of the tumour by a research society,

who reported it to be a glioma of the retina, which did not satisfy me. I therefore submitted the section to Mr. R. Affleck Greeves, who kindly made the following report on it :

Description of a microscopical section, taken across the posterior part of the eyeball at the level of the optic nerve.—A large nodule of growth is seen—this is evidently the projecting part of a mushroom-shaped mass, which has been cut across beyond the level of the place of origin of the main mass from the choroid. A double layer of retina surrounds the nodule, showing that a considerable detachment was present. The growth is undoubtedly choroidal in origin, and consists of a mass of cells which show no sign of any special mode of arrangement.

Fine layers of fibrous stroma traverse the mass. Numerous blood-vessels are present—these consist of cleft-like spaces, each lined by a single layer of endothelium. The cells which make up the growth are, for the most part, of moderate size—they contain well-marked nuclei, which stain deeply with hæmatoxylin, and enclose distinct nucleoli—the latter are frequently two in number in a single nucleus.

Both round cells and spindle cells are present—their relative proportions vary in different parts of the growth.

No pigment is to be found in any part which appears in the section.

The growth is a fairly typical leuco-sarcoma of the choroid.

The occurrence of two different types of malignant disease in the same patient within a year, is, I think, unusual ; either growth, unless removed in time, would have had a fatal result, but, so far, she still enjoys good health. I do not think there is any connection between the epithelioma of the lip and the sarcoma of the choroid, except the mere coincidence of their occurring in the same patient. No relative of hers, that she knows of, has had any kind of growth, or been operated on for a growth in the eye or elsewhere.

X. DISEASES OF THE RETINA AND OPTIC NERVE.

1. *A case of detachment of the retina at the ora serrata.*

By A. Lawson.

(With Plate VII, fig. 4.)

Clinical reports of detachment at the ora serrata must be very rare. In my somewhat hasty search through ophthalmic records I have not managed to find a single case, and certainly I have never seen a similar case to this. As a pathological specimen the condition, though rare, may occur if there is a great shrinkage of inflammatory vitreous exudates, but in such cases the eye must generally be blind before the occurrence, or, any rate, so nearly so that a detachment at the ora serrata could not be seen. In the case I am about to relate the diagnosis was made clinically by ophthalmoscopic examination and subsequently confirmed by pathological investigation.

The patient was a young lieutenant, æt. 23 years, who came to see me first at the latter end of October, 1915. He had been wounded at the Dardanelles in the previous May. A bomb burst near him, wounding him in the right groin, and almost simultaneously he was struck on the left leg by two bullets, one of which killed his neighbour on his left. Please observe that he was not to his knowledge wounded in either eye, but he said that his neighbour on the left was shot in the head and that the head was greatly shattered and that he was struck and spattered on the left side of his face by fragments of the skull and soft parts, blood, etc.

When he came to me the condition was as follows :

Right eye.—Vision = $\frac{6}{5}$ easily. Eye normal in every respect.

Left eye.—Vision = $\frac{6}{24}$, not improved. The pupil was dilated to its widest limits, and immobile. There was a slight general vitreous haze with one rather large grey opacity which moved freely. The condition of the retina and choroid was perfectly normal. The movements of the eye were free and painless, the sclera was quite white, there was no sign of corneal precipitates, and the pupil was evenly dilated without notching or any sign of inflammatory adhesions.

In fact, the eye was perfectly quiet.

Examination of the field showed a slight general and fairly even contraction. Centrally there was a small relative colour scotoma.

I concluded that he was suffering from the effects of a severe concussion. This view was favoured by the history and by the fact that the face on the left side was a good deal bruised at the time he was wounded.

I told him that the length of time that had elapsed since the accident made the prognosis as to recovery of sight rather doubtful, but I thought there was a great probability that he would not get any worse.

He was not much inconvenienced except that he was happier with the left eye covered. This I put down as being in part, at any rate, due to the mydriasis, and I ordered weak eserine drops (1 gr. ad. 1 oz.) to use occasionally.

I saw him two days later and the eserine had answered perfectly, contracting the pupil to its fullest extent, and his vision improved thereby to $\frac{6}{12}$ and letters of $\frac{6}{9}$. I might say that I could not get this improvement with a stenopœic disc on the previous visit.

He was told to go on using the eserine and I reduced the strength to $\frac{1}{2}$ gr. ad. 1 oz.

I saw him again about six weeks later. The eserine had been only successful in that it shut down the pupil

for a few hours. If not used, the mydriasis became as extreme once more as it ever had been. The attempt to improve the action of the sphincter by stimulating it had therefore failed entirely. Further, the eserine caused a good deal of temporary aching and confusion of sight after its use. On this account I advised the patient to give it up.

The vision at this date was precisely as before, and the field and ophthalmoscope picture unchanged.

I next heard of him by a wire on February 1st, saying that he had suddenly lost a great deal of his sight. When I saw him he told me that when out walking with a friend he felt the sight of his left eye become suddenly worse, so that about three-quarters of the whole field disappeared and he could only distinguish objects placed high above the horizontal level. When he got home he noticed that the pupil, which was widely dilated as usual on starting out, had become much less.

On examination I found the pupil equal in size to the other and reacting both directly and consensually though not normally so. The eye was rather photophobic and flushed and the vision reduced to hand movements. I dilated the pupil and an extraordinary picture was revealed. A fine lace-like veiling, which focussing showed to be the retina, was hanging detached over the lower two-thirds of the picture, leaving a gap above it through which the choroid and its vessels were easily and plainly seen. The upper margin of the detachment went straight across the field and was quite close to the lens and quite uniform in outline and sharply marked without apparent rent or fold. I concluded that it must be a detachment very close to the ora serrata and ordered him to bed in a nursing home. At the same time, seeing that the picture was so strange and unusual, I asked Mr. Treacher Collins to come and see the case with me. The occurrence of this detachment and the consecutive change in the pupil made me suspect that there was after all a foreign body in the globe somewhere about the ciliary region, the

irritation of which had induced the pupil changes and which in some way difficult to explain had brought about the detachment. Consequently, after a few days' rest in bed, during which time all the photophobic and inflammatory symptoms disappeared, I asked Sir Mackenzie Davidson to skiagraph the eye. This he did, and found a minute foreign body localised in the neighbourhood of the ciliary body on the outer side. I decided that considering all the circumstances there was nothing to be gained by attempting to remove it.

After a short stay in the nursing home, during which time the upper field became defective also, I sent him home to rest quietly for a time: when at home the lower visual field became much better, so that he could once more distinguish large objects below the horizontal line, but the upper field remained very bad.

When seen again, about four weeks after leaving the nursing home, I found that the retina to a large extent had apparently re-attached itself above, and I could for the first time make out a small shining foreign body high up on the outer side and just within range of the ophthalmoscopic examination.

The eye soon afterwards began to show signs of disorganisation and became very irritable, the tension became markedly hypernormal and the iris became more and more discoloured, which I thought was probably due to siderosis. He was also getting pain at times and, all things considered, I strongly advised him to submit to enucleation, which was performed about three months after the first appearance of the detachment. I may add that the foreign body disappeared from view shortly before I removed the eye. It apparently shifted up with the retina in which it was entangled.

Pathological Report by E. Treacher Collins.

Eyeball received from Mr. Arnold Lawson. It had evidently been in spirit, as the cornea was opaque. It

was transferred to a 4 per cent. formalin solution and afterwards frozen and divided by an antero-posterior vertical section. The cornea, besides being opaque, was somewhat wrinkled, due to hardening re-agents. The lens was opaque due to the same cause. The anterior chamber was filled with a gelatinous coagulum. The lens was rounder in shape than normal and displaced slightly upwards. The retina was detached and formed a much puckered membrane lying in the centre of the eyeball. There was hardly any vitreous chamber left, only a small area posteriorly. The space between the retina and choroid was completely filled with a grey coagulum of gelatinous consistency. After this had been removed from the outer half of the globe, the retina at the ora serrata, together with a portion of the pars ciliaris retinæ, was found to be torn away from the ciliary body, both above and below. The only portion of the retina in the region of the ora serrata which retained its attachment was on the outer side of the globe. The whole circle of the pars ciliaris retinæ together with the suspensory ligament of the lens had become drawn towards the centre of the globe and to its outer side. At the outer and upper part of the globe a conical projection was seen to pass from the outer surface of the detached and puckered retina, and pass backwards to become adherent to the choroid a little in front of the equator. At the spot where the retina was adherent to the choroid there was a circular area of choroidal atrophy. Behind the lens, at its lower margin, between it and the detached pars ciliaris retinæ, a small foreign body was found, which on being touched crumbled into two. It was attracted by a magnet.

The spot of choroidal atrophy in the upper and outer part of the globe, to which the retina was adherent, was evidently the point of entrance of the foreign body. It must have passed through the vitreous behind the lens to the ciliary region on the opposite side of the globe. The contraction caused in the anterior part of the vitreous, in

the track of the wound, was the probable cause of the dragging inwards towards the centre of the globe of the hinder portion of the pars ciliaris retinæ and the retina in the region of the ora serrata.

The President said he was allowed, through Mr. Lawson's kindness, to see this case clinically. It was unlike anything he had previously seen. It looked as if the retina had been dragged down from above so that one could see the choroid exposed over a large area. He had examined eyes pathologically in which there had been concussion injuries and in which he had found the retina detached from the ora serrata, though the appearance could not have been seen clinically owing to opacity in the lens. At Moorfields Hospital Museum there were two or three specimens in which the retina after blows on the eye had been torn away at the ora serrata.

Mr. J. Herbert Parsons desired to mention a case he saw last year which was admitted to Moorfield's Hospital, and was seen by several members of the staff there. The patient was a girl, æt. 17 years, who was brought to him with the history that in March, 1915, she had an injury to the left eye. She saw Mr. Collier Green, of Derby, who wrote to him afterwards, giving details of what he found, namely, a small rupture of the choroid at the usual situation. She came to him, the speaker, with the statement that the sight in the other eye had been failing for four weeks. He looked into the right eye—the one of which she complained—and found she had detachment of the retina outward, with a very distinct edge, beyond which one looked straight on to the choroid. In the left eye there was the choroidal rupture, and a detachment of retina in the lower part of the eye, with exactly the same appearance as in the right eye. By getting the pupil dilated to the extreme extent, even then one could not see beyond the apparent hole in the retina. He believed that in both eyes the detachment was from the ora serrata, but no certain proof could be given.

He thought it possible that in these cases the detachment occurred at the ora serrata—certainly in the concussion cases—as the result of cystic degeneration of the retina in that position. This was a common occurrence in old people, and it was a not infrequent occurrence in cases of concussion. It was easy to conceive that if the condition extended further, the retina might be ruptured, in the same manner as Coats proved conclusively that actual holes at the macula occurred as a result of a similar process of cystic degeneration. Still, it was very difficult to understand why this girl got an exactly similar type of detachment, which he had never previously seen, in an eye which had not been injured. Possibly it pointed to a similar abnormal condition at the ora serrata, a condition which was the precursor of the detachment.

Mr. Harrison Butler said that a month ago he removed an eye from a man who had received a blow on it from the bough of a tree. He removed the eye because it was septic. When he bisected the eye, he found a gap between the ora serrata and the edge of the retina, over one-third of the circumference. There was a perforating wound, and probably the part was torn away by the blow. It was not, however, a deep perforation.

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2. *Symmetrical pigmented changes at the macula.*

By F. A. C. Tyrrell.

I. L—, only child, æt. 9 years. Father and mother living and healthy. No history of blindness or mental defect on either side. Mother one of seventeen. Patient has always had good health, and is of normal mentality. She first came under my notice in February, 1916. Her vision was then $\frac{6}{60}$ and J. 8. R. and L., and has not appreciably altered since. The right eye is divergent. The ophthalmoscopic appearances are symmetrical. A small circular

area of densely-powdered pigmentation is seen in the macular region of each eye, surrounded by a ring of rather more scattered pigment spots. The whole fundus is lightly peppered, the discs are slightly on the pale side. Both eyes are emmetropic. The condition has not varied during the period in which she has been under observation.

The child was of normal mentality, and there was no history of disease in the family. The mother was one of a family of seventeen, all healthy. This was, therefore, separate from amaurotic family disease, which was sometimes discussed in connection with these cases of symmetrical pigmentation. He had had the case under observation eighteen months, and there had been no change during that time. The pigmentation was chiefly in the macular region, in both eyes, but there were scattered spots in different parts of the fundus, and some slight choroidal degeneration. The discs were slightly pale.

Dr. G. Mackay said the description of this case reminded him of a child he saw many years ago, in whom there was a gumma on the tibia.

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3. *Fundus lesions in cases of war nephritis.*

By J. Jameson Evans.

The Medical Research Committee is investigating selected cases of war nephritis, and among other particulars concerning which the Committee desires information, is the state of the fundus oculi. In the course of my examination of these cases at the First and Second Birmingham War Hospitals I found certain fundus changes which suggested to me that if ophthalmoscopic investigations were to be of any help to the Committee, we should

have to examine *every* case of war nephritis, and the results obtained by different observers should be then examined and correlated in order, if possible, to decide whether this particular form of nephritis causes or is associated with changes in the fundus; how far these changes may be regarded as of renal origin and whether they can be regarded as of any diagnostic or prognostic significance. Such a wide investigation can only be carried out by the Research Committee with the help of all the officers who have charge of these cases of nephritis, but I think their labours will be advanced if ophthalmic surgeons will provide detailed reports in so far as their speciality is concerned.

I would like to mention that so far as my experience goes, the fundus changes which are likely to be met with are very slight in degree and may easily be overlooked unless the examination be made after dilatation of the pupil and by direct ophthalmoscopy. Another point I would like to impress on you is that I do not wish to imply that these changes are the result of trench or war nephritis—that can only be decided when we have the collected reports of all the investigators—and they must only be accepted as lesions which have been observed *in* cases which have been diagnosed for me as war nephritis. Further, no attempt has been made to differentiate types or degrees of nephritis, but I think that in future some effort should be made in this direction, but this again would have to be done through the Research Committee and the officers in charge of the cases. The following is the result of my examination of the fundus in 100 cases of war nephritis:

Normal fundus . . . 43 per cent.
Changes noted in the fundus . 57 „

The fundus changes observed:

Involved the optic nerve and retina in thirty-nine cases.

Involved the choroid in thirty-four cases.

(1) *Lesions of the optic nerve and retina.*—These were

in the main very insignificant, and some were so slight in degree that they were doubtfully pathological. In twenty-two cases the optic disc was hyperæmic and slightly œdematous; in thirteen cases there was peri-papillary retinal œdema, some perivascular lining, and tortuosity and fullness of the veins. In eight cases the disc appeared pale or greyish without any definite atrophy of the nerve-head. In only one case was the classical appearance of the hæmorrhagic neuro-retinitis of albuminuria observed, and this was in the case of a man who was æt. 56 years, and who evidently suffered from chronic Bright's disease. In no other case were retinal hæmorrhages observed. In no other case was any definite evidence of arterio-sclerosis noted, although in two cases there was some suspicion of arterial pressure on the veins. Fine dotted changes in the retina in the macular region were observed in four cases, and one case showed some œdema at the macula with reduction of vision to $\frac{6}{24}$.

(2) *Changes in the choroid.*—I was rather surprised to find so many cases showing evidence of choroidal disturbances. Here, again, doubts could be entertained as to their pathological character. Particularly does this apply to eighteen cases which showed depigmentary change in the equatorial region. These depigmented areas were sometimes arranged diffusedly around the periphery and often showed fine pigmentary deposits; sometimes these changes were confined to a section of the globe, at other times they appeared as whitish or yellowish streaks with slightly pigmented borders reaching from the intermediate zone to the periphery. In none of these cases was there any evidence that there had been any acute choroiditis. In twelve cases, however, there were discrete orange-coloured spots with little or no pigmentation, but evidently of the type of disseminated choroiditis. Two cases showed typical areolar choroiditis with marked pigmentation such as one might ascribe to syphilis.

Five cases showed guttate changes in the macular region, and these we should at once diagnose in patients of

more advanced age as central guttate choroiditis or Tay's choroiditis.

The most peculiar choroidal changes observed were patches of slate-grey discoloration in the neighbourhood of the macula. These spots—only one of which was larger in size than the optic disc—appeared to be slightly raised and the retina over them gave a sheeny reflex. There were five of such cases. In three the spots were situated along the inferior temporal vessels, one along the superior temporal, and one on the outer edge of the macula. They seemed to be deeply situated, and only in the last case was vision affected ; and it is doubtful if even in this case the depreciation could be ascribed to the slatey spot, as the vision was the same in the unaffected eye ($\frac{6}{12}$). It may here be remarked that except where the macula was œdematous there was no reduction of vision, and in no case was I asked to examine a patient because he complained of his sight.

Assuming that the fundus changes observed were due to war nephritis, it might be suggested that the character of these lesions might throw some light upon the nature of the poison which produces them.

As to the *character of the lesion* it would appear that both in the retina and the choroid the process is of a low inflammatory nature, giving rise on the one hand to slight œdema of the nerve-head and neighbouring retina and occasionally a few fine retinal deposits ; on the other, setting up depigmentary changes in the hexagonal epithelium and choroid, either with or without slight and fine pigmentary deposits and occasionally more acute but more localised foci showing as fine areolar spots, with very limited pigmentary deposits at their edges. The more definitely punched-out yellow or white spots with markedly pigmented rims have been ascribed to syphilis.

The slatey patches are suggestive of small choroidal hæmorrhages, but I can hardly believe they are of that nature, seeing that no hæmorrhages were observed in the retina and there was no evidence of choroidal congestion. It is possible they were due to small choroidal thromboses.

The nature of the poison.—The changes observed seem to point to a toxic or infective origin. They approach in the main the fundus lesions we associate with the toxicity of advancing age or senility. Diffuse depigmentary changes I have also observed in children and have ascribed them to the auto-toxæmia of gastro-enteritis and such internal disturbances as infants are addicted to. The nephritis is of the type which follows scarlet fever, and it is possible that the changes are produced by an infective element. It is also possible that fundus lesions are not uncommon in scarlatinal nephritis, but few of us have had the occasion to examine many cases of the kind. Syphilis has been suggested as the cause, but except in the two exceptional cases referred to above, the appearances of the fundus changes were not, to me, suggestive of syphilis.

Sepsis, particularly oral sepsis, has also been suggested as a cause of the nephritis, but there is nothing in fundus lesions any more than in the epidemiological or bacteriological aspects of the disease that gives any support to such a suggestion.

4. *Hereditary glioma of the retina.*

By A. Hill Griffith.

The personal experience on which this paper is based is comprised in the account of two families under my charge some years ago at the Royal Eye Hospital in which the disease was transmitted from the mother to several of her children.

I shall give the particulars later.

At the time, eleven and fourteen years ago, when these patients were seen by me, neither I nor any of my colleagues had seen any cases of hereditary glioma, and I was myself unaware of such cases having been put on record.

I do not think that I could have passed over any such cases, and for the following reason; I have never seen a child with glioma that was not brought by its mother, and if she had lost an eye, even if wearing an artificial eye, I could not have failed to note this and make inquiries as to how the eye was lost.

To my mind this is quite conclusive as far as transmission from the mother is concerned, but in transmission from the father, and we shall see that this may take place, it is quite possible that we have passed over some such cases, as the father being at work might not in every instance come with the mother and child.

Another possible source of error is exemplified in a case mentioned by Berrisford, where a man, Thomas Grover, had one eye removed for glioma and his daughter had four children all with glioma, three of them bilateral, although she herself was free from this affection, the disease skipping one generation.

Making all allowance for these factors I think we must admit that transmitted glioma is a rare occurrence and that any cases should be put on record; and I hope that members will give us the benefit of their experience.

The particulars of my two gliomatous families are as follows:

The Smith family.

(1) John. First seen by me January 3rd, 1903, when æt. 5 months. The left eye was removed for glioma a few days later. On September 23rd, the mother thought the right eye was affected, and a flocculent-like mass could be seen at the nasal side of the fundus after dilating the pupil. This eye was also removed in October of same year.

The mother, æt. 22 years, had her right eye removed by the late Dr. Mules at the age of nine months for a growth, and *her* mother, who is still alive, tells me that her daughter's eye was exactly like those of her grandchild's and like them "flashed fire," so I imagine that there can be no doubt as to the nature of the affection, although I failed to find a record in the hospital books.

The child kept well till the age of 7 years, when a tumour developed in the right upper jaw and palate, for which an operation was done at another hospital; the child died some three weeks later and examination showed secondary growths in both kidneys but no growth within the skull, only an œdematous state of the brain. I cannot say from the information available whether the growth was from the glioma of the eyes or not.

(2) Ethel. Now æt. 12 years. Both eyes are normal in every way.

(3) Lucy. First seen November 10th, 1906, when æt. 9 months. The mother noted the growth in the left eye three months ago and for the last week also in the other eye also. Both eyes were removed and the child died on July 5th, 1907, from an attack of measles.

(4) William. First seen April 8th, 1908, when æt. 9 months.

Both eyes were removed for glioma and he died of this disease at the age of 2 years and 11 months.

(5) Dead born.

(6) Mary Elizabeth. First seen April 26th, 1913, when æt. 1 week.

Examination of eyes at this time revealed no glioma but five months later it showed in the left eye which was removed, and shortly after the other eye was seen to be affected, but it was not removed and the child died of broncho-pneumonia eight months after this.

(7) I saw the mother the other day and she has a baby æt. 10 weeks, which so far shows no signs of the disease, but in view of the fact that I could not make a very satisfactory examination of the eyes, it is quite possible that the disease is already present or, as in the case of Mary Elizabeth, may come on later.

The mother tells me that Ethel and the baby were the only ones that were bottle-fed and she ascribes their immunity to this fact; I do not know whether she is correct, but her opinion is as good as my own, which I therefore need not express.

Here, then, we have a family of six children, not counting the still-birth, with double glioma in four.

The Jones family.

(1) Still-born, at present date would have been æt. 14 years.

(2) Doris. First seen October 3rd, 1906, when æt. 3 years.

The right eye was removed for glioma. The child, now æt. 13 years, is well and the other eye is normal.

The mother had her right eye removed for glioma by Dr. Glascott in July, 1879, at the age of two and a half years, and I show you the hospital paper with the above diagnosis in Dr. Glascott's own handwriting.

(3) George. Seen December 7th, 1907, æt. 3 years. The right eye was removed for glioma and at the present date the child is well and the other eye is normal, ten years after operation.

(4) Florence. Seen November 19th, 1913, when æt. 7 months. The right eye was removed for glioma and the left also for same affection at age of 1 year and 10 months. She is now well and in good health.

All the children were breast-fed.

Here we have three children, not counting the still-birth, with glioma of both eyes in one, and single glioma in the other two children.

As I am concerned with the factor of heredity only, I refrain from giving any description of the eyes either before or after removal, and need only say that in no case could there be the faintest doubt as to the nature of the affection; indeed, they were all typical examples of the disease.

In compiling the appended list of references I have been greatly assisted by Col. R. H. Elliot and Mr. Sydney Stephenson and beg to tender my best thanks to these gentlemen.

As far as I have been able to make out there are five cases of hereditary glioma of the retina on record, which with my own two brings the total to seven examples.

In two instances only, Owen's quoted by Berrisford and others, and de Gouvea's was the transmission from the father, but for the reason already mentioned this may not be so important as it appears.

References.

(1) Hoffman.—"Hereditary Bilateral Glioma of the Retina," *Trans. 25th Ophth. Congress Heidelberg.*

(2) Lukens.—"Glioma of the Retina," *Journ. of Ophth. and Oto-Laryng.*, January, 1908.

(3) De Gouvea.—"Hereditary Retinal Glioma," *Ann. D'oc.*, 143, p. 32; *Ophthalmoscope*, vol. viii, p. 531.

(4) Caspar.—"Inheritated Retinal Glioma," *Cent. f. Augenheilk.*, vol. viii, p. 223; *Ophthalmoscope*, vol. viii, p. 289.

(5) Dabney.—"Intra-ocular Neoplasms," *Amer. Journ. of Surgery*, vol. xxix, p. 185, 1915.

(6) Berrisford, Paul D.—"Statistical Notes on Glioma Retinæ with a Report of 41 Cases," *Royal London Ophth. Hosp. Reports*, vol, xx, pt. ii, March, 1916.

The President said that the occurrence of such cases as Mr. Hill Griffith had dealt with was very rare. At Moorfields the records of glioma cases had been kept for many years, and out of 135 such cases there was not a single one in which a second member of the family was known to be affected. He thought one reason why inherited glioma was so seldom met with was, that so few patients affected survived until the age at which children were born.

Mr. Johnson Taylor said he was able to speak of one case. Many years ago he removed the eye of a child whose mother had her eye removed when she was æt. 9 months, the presumptive evidence being that it was glioma.

Mr. Priestley Smith (Birmingham) suggested that the enquiry which Mr. Hill Griffith proposed should be ex-

tended a little. One of the theories of the causation of glioma was that the cells were of the nature of undeveloped fœtal cells, and if so, they might be regarded as cells of imperfect development. Hence it might be well for investigators to look for other signs of arrested development.

Mr. R. R. Cruise said that about ten years ago, at St. Mary's Hospital, a child had both eyes removed for glioma, a second child also had to be similarly treated, while a third child was being brought regularly because the mother was very anxious about the fate of the second. He believed there was a record of that family in one of the volumes of the Society's *Transactions*.

Mr. Hill Griffith (in reply) thanked Mr. Cruise for mentioning the case he had.

With regard to Mr. Priestley Smith's remark, he, the speaker, had never been struck, in these cases of glioma, with any other indications of backward development in other respects in these children. He had always been in the habit of telling students that these children looked particularly well; they seemed to be even more chubby than the generality of young patients.

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5. *Two cases of Leber's disease (hereditary optic atrophy), with X-ray examination of the sella turcica.*

By W. B. Inglis Pollock.

The following brother and sister were sent to the Glasgow Eye Infirmary by the school doctor on account of defective vision. They were admitted to Dr. Leslie Buchanan's Clinic, but through his kindness they have been under my care since then. As the sister is the older, her case is related first.

A. R—, æt. 11 years, was first seen on October 9th,

1915, when she was put on atropine for a fortnight without her vision being tested, as the infirmary is so understaffed owing to the war. On October 23rd, both optic nerves were found to be slightly congested. Retinoscopy

R. and L. $\left|\begin{array}{c} +4 \\ - \\ \end{array}\right.$ + 5, V.R. and L. with + 1·5 D. sph.

and + 1 D. cyl. axis 90°, $\frac{6}{24}$. By December 4th the optic nerves were seen to be paler, and she was put on hyd. c. cret. 1 gr., with ferri. pot. tart., 1 gr. This treatment was continued until October, 1916, during which time she was brought to the infirmary at intervals of four to six weeks. The vision became steadily worse. By April 15th, 1916, V.R. fingers at one foot, V.L. $\frac{3}{60}$; and on September 2nd, 1916, V.R. and V.L. hand movements.

By this date, Mr. Fisher's paper on Leber's disease (1) had appeared in the August number of *The Ophthalmoscope*, and I, therefore, arranged for an X-ray examination of the sella turcica. This was kindly done for me early in October, 1916, by Dr. D. O. MacGregor, Medical Superintendent, Victoria Infirmary, Glasgow. The plate showed a shadow like a very small bean, with the concavity downwards, and situated in, but a little below, the centre of the sella turcica. The print does not show it so distinctly, although it can be seen. The negative and print from her brother gave the same appearance. The treatment was immediately changed from mercury to hypodermic injections of Messrs. Parke, Davis and Co.'s preparation of pituitrin, and thyroid extract by the mouth, with instructions to Dr. Wilson, of Irvine, for increasing both until signs of tremor appeared.

On February 3rd, 1917, V.R. and V.L. $\frac{4}{60}$. For some reason the treatment was apparently stopped for a month, and by March 27th V.R. $\frac{1}{60}$, V.L. fingers moving. The treatment has been resumed, and on April 24th, 1917, V.R. $\frac{1}{60}$, V.L. fingers at 6 in. She is now receiving an injection of 2·25 c.c. pituitrin twice a week, and 16 gr. of thyroid extract daily.

The optic nerves are as seen on the last-mentioned date, pale in the external half, with a shallow cup, not filled with connective tissue. The fundus is otherwise normal. A slight tendency to nystagmus appeared two months ago, but it is not now present.

The field of vision on March 27th, 1917, showed a central scotoma, with general shrinking of the peripheral field. The scotoma could not be mapped out.

F. R—, æt. 8 years, was first seen on August 28th, 1915, and ordered atropine. A week later, retinoscopy R. and L. $\left|\begin{matrix} \\ \\ \end{matrix}\right.$ $\begin{matrix} -0{\cdot}5 \\ \\ +\ 1{\cdot}5 \end{matrix}$. Ordered + 1·25 D. cyl. axis 90. The fundus appeared normal, and the vision was not taken.

October 9th, 1915.—V.R. with his glasses $\frac{2}{60}$, V.L. $\frac{6}{36}$. The optic nerves were slightly congested, and he was directed to persevere with the atropine, and to be kept from school.

December 4th, 1915.—Both optic nerves looking pale, and a slight swelling of 1 D. He was given the same powders of mercury and iron as his sister.

April 15th, 1916.—V.R. P.L., V.L. fingers 3 ft.

September 2nd, 1916.—Vision as in April. An X-ray examination was undertaken at the same time as his sister's case by Dr. MacGregor. The plate shows the same appearance as in her case in the sella turcica. The print is not so distinct. He was put upon the same treatment as his sister, and is now receiving 1·75 c.c. pituitrin, and 12 gr. of thyroid extract daily by the mouth.

February 3rd, 1917.—V.R. hand movements, V.L. fingers 2 ft. The optic nerves are pale and muddy, and the vessels narrow.

The field of vision was not tested until March 27th, 1917, when it was found to have a central scotoma and some shrinking of the periphery; still, it was difficult to be sure of these data.

Family history.—The mother has a strabismus divergens

of the left eye, with myopia of 30 D. The fundus is normal, apart from a myopic crescent. Her right eye has $\frac{6}{6}$, with a normal fundus. The father is said to have normal vision. The mother stated that she had had two miscarriages and a stillborn child before the daughter was born. The son followed, and later another miscarriage occurred. There are thus only two children in the family. No history of blindness in other relatives was elicited. The only brother and sister of the mother died of tuberculosis of the lungs.

The treatment by mercury was instituted as the result of obtaining the above information. It was continued for forty-six weeks, or nearly eleven months, during which time the vision of both children became steadily worse. The mercury was abandoned on October 21st, 1916. The mother and the two children were sent to Dr. J. A. Campbell, bacteriologist, Glasgow Royal Infirmary, April 30th, 1917, or six months later, for Wassermann reaction. He finds all three to be clearly negative, using the Boas technique with the modified antigen of Sachs, although he himself expected to have the opposite result. The mother has not had mercury, and the period of six months excludes any influence from it on the test in the case of the children. The children have never had salvarsan, or any of its derivatives.

The diagnosis of Leber's disease is based on the condition of the optic nerves, the absence of all signs of hereditary syphilis, the age of onset, the character of the fields of vision, although they were perhaps uncertain, the relationship of the two children, the almost complete loss of vision under anti-syphilitic treatment, and finally the relative improvement after the treatment with organotherapy.

X-ray photographs were again kindly taken by Dr. MacGregor on April 26th, 1917, when the same appearances were found in the sella turcica as in the plates of October last.

The shadow found in the sella turcica resembles a small

kidney-bean with the concavity downwards. There is just a faint suggestion of threads passing from four corners of it diagonally towards the walls of the sella. Dr. MacGregor has never seen anything like it, nor have I myself. He considers the appearance suggests something pathological. No honeycomb-like appearance was observed as in Mr. Fisher's case.

The treatment with organo-therapy was probably commenced too late to be efficient. The girl's vision has risen from hand movements to $\frac{1}{60}$ and counting fingers, while the boy has improved in his right from perception of light to counting fingers, and in the left increased the distance for counting fingers. The organo-therapy is being continued, and the pituitrin raised to 3 c.c. for the sister, and 2 c.c. for the boy.

Reference.

(1) Fisher, J. Herbert.—*Trans. Ophth. Soc. U.K.*, vol. xxxvi, p. 298.

6. *A further case of Leber's disease, and two allied cases, associated with changes in the sella turcica.*

By J. Herbert Fisher.

You will allow me, Mr. President, to thank you and the Society for having deferred this paper until I could be present. It is a subject I am much interested in, having been the first to suggest that Leber's disease was probably to be attributed to an over-activity with enlargement of the pituitary body, that over-activity being associated either with the period of adolescence, or, in the female sex, with the period of sexual decline. It was my privilege to bring forward my evidence in favour

of this view at the last meeting of this congress. I am interested in Mr. Inglis Pollock's cases, and I sympathise with him in the difficulty he has in demonstrating before a meeting changes which can be detected in the radiographic negatives and in the prints obtained from them. It is very difficult, when projecting them on to the screen and when handing them round, to get men to see the changes which are readily visible when pointed out under the best conditions. I found that difficulty last year, and I have the same sort of difficulty in bringing forward one or two other cases.

The only example which could be spoken of as one of familial hereditary optic atrophy to which I have to allude to-night, occurred in three cases—two brothers and an uncle.

Pte. H. G—, æt. 21 years, on service in France, had always had good sight up till August, 1916. At the beginning of this month his vision began to fail, and on October 12th he reported himself sick in consequence. After eight days' observation in a local hospital he was returned to duty, but, a fortnight later, was sent to another hospital for the same cause. In every other respect, except for the failure of vision, he was well and healthy. He was admitted at Moorfields Hospital on November 11th, 1916, when he could barely count fingers at a foot with either eye. He had been smoking daily about twelve cigarettes and two ounces of tobacco per week. Patient says he sees better on a clear night, and under this condition sees objects much more distinctly than by daylight. He counts fingers better with the peripheral portion of the field than by central fixation, and, as regards colours, can see no colour even with distant objects straight in front of the eyes, and with the peripheral field makes mistakes as far as red and green are concerned, though he names blue and orange correctly. At first, there was no very definite change apparent in the optic discs, though there was some difference between the two, but, by the latter part of November, I was quite satisfied that

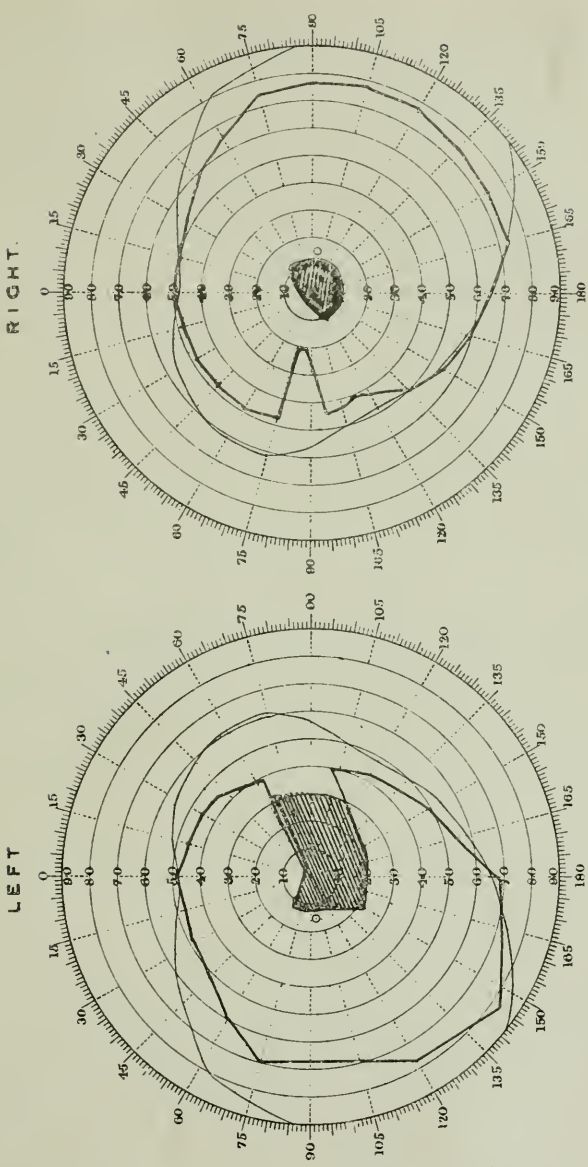


FIG. 26.

certainly in the right eye, and probably in the left, there was pathological pallor in the lower outer portion of the disc. The fields of vision were charted on November 12th, and are as shown.

On December 6th, 1916, between 6 and 7 p.m., 150 grm. of glucose were administered, and at 9 a.m. on December 7th a specimen of urine contained no sugar; there was no abnormality in regard to the quantity of urine passed. An X-ray picture of the sella turcica was obtained, and shows a definite, though not very extensive expansion, undermining and irregularity in the definition and shape of the posterior clinoid processes.

I learnt that his brother, Fred G—, æt. 22 years, was a patient of Mr. Percy Flemming at University College Hospital, and he kindly enabled me to see him. The history given by F. G—was that in July, 1916, he could see as well as he ever did, and as well as anybody else, but that early in September, 1916, he first noticed a mistiness of distant sight. The left eye is, and has been throughout, the worse of the two. He had been attending at University College Hospital since November 6th, 1916. The vision of his right eye when I saw him on November 22nd, 1916, was $\frac{1}{60}$. There was no material error of refraction in either eye. In the right eye there was an old patch of choroido-retinitis just above and inside the macula. The vision of the left was the same as that of the right eye. The pupils acted normally both to light and on convergence. The optic discs were not by this time obviously atrophic. Veins were a little tortuous, the fields were as attached, uncontracted. Like his brother, he saw better on clear star-light nights than by daylight. Using colour cards about two inches by three he named red and yellow incorrectly at the first attempt, but recognised blue and green, and himself appreciated that he saw red and yellow much more brightly in the peripheral field.

He stated that in January, 1916, he passed his army test and read $\frac{6}{6}$ with either eye. This patient was also

X-rayed at Moorfields Hospital, and the sella turcica outline was found to be slightly indistinct posteriorly, but it was not elarged or distorted, and, personally, I should have considered it as normal. The fields of vision were not contracted.

I learnt that an uncle of the above two patients, Thomas V—, a baker by occupation, was a patient of Mr. Lang at Moorfields Hospital when æt. 21 years. The out-patient letter of Thomas V—, dated June 18th, 1896, was produced. It was noted that the vision in each eye was the ability to count fingers at a metre, and to read J. 16. Both pupils reacted sluggishly, and the disease was regarded as optic neuritis, passing on to atrophy. A biniodide mixture was ordered. July 6th, 1896, optic discs were white and woolly, outlines very hazy, vessels about normal in size, but very tortuous. Connective-tissue on the disc and extending away from the disc for a short distance. He remained under observation until April, 1897.

The vision appears to have become slightly worse, the left eye being rather worse than the right, J. 19 being puzzled out with difficulty by this eye.

In March, 1897, the right disc margin fairly clear, normal vessels, not tortuous, very pale. Left disc margin still blurred, some vessels, especially upwards, keep tortuous, also getting very pale.

These three patients, all males, the uncle being a brother of the mother of the two younger patients, form a typical group of Leber's disease, and in one, my own, there was certainly some altered condition of the sella turcica. A Wassermann test was employed in the case of this patient, and was negative. The implication of the optic nerves in each of these three patients, all of whom were males, occurred in two instances at 21, and in the third at 22 years of age.

The cases are reported in support of the view which the author first suggested a year ago that Leber's disease is due to an exaggeration of the activity of the pituitary

body correlated with the sexual function, whereby the hypophysis becomes sufficiently enlarged to implicate the visual pathways by direct pressure or stretching; such change in the gland can, in some cases, be demonstrated by deformation of the sella turcica or by shadows thrown by the substance of the gland, and the effects of organotherapy on Leber's disease should be studied and diligently pursued in recent cases.

Two cases allied to Leber's disease, but without family tendency.

Case 1.—Lily S—, a girl, æt. 13½ years, was an inpatient under my care at St. Thomas's Hospital from February 17th to April 2nd, 1916. She was the eldest of six living children; two had died of wasting.

Her mother had had no miscarriages. The vision had been defective for the previous three or four months, during which time she had suffered from headaches in the frontal area, but there had been no vomiting.

There was no family history of failure of sight obtained in any direction. The patient had once suffered from rheumatic fever and the apex-beat of the heart was in the fifth space, an inch outside the nipple line—there was a blowing, systolic murmur heard at the apex and conducted out to the axilla.

The vision of the right eye was $\frac{3}{60}$ and of the left $\frac{5}{60}$. The pupils were semi-dilated, and though each responded to light, the contraction was very indifferently maintained. Each optic disc was pale, that of the right eye being the paler, and in each the temporal side showed more loss of colour than the nasal side. The pallor at first was but very slight, but gradually became undoubtedly increased and is by now very striking.

An examination of the nose and its accessory air sinuses, by Mr. Howarth, showed no disease of any kind.

The Wassermann reaction was negative. Menstruation had not been established, but has since developed rather later than usual at the age of 14 years and 8 months.

The patient stated that sight first began to fail in October, 1915, about a week after Zeppelin bombs were dropped in the vicinity of Liverpool Street, near which she lives. She thought that both eyes were affected about the same time, but they became slowly worse. She, however, continued to go to school until a few weeks before she was admitted to the hospital, but she found difficulty in doing her work, apparently on account of loss of sight and headache. An X-ray photograph of the pituitary fossa showed nothing abnormal. The fields of vision were charted, and cannot be said to be abnormal, though in the right there is more loss on the temporal side than in any other position, when tested with red test objects. After leaving the hospital she continued attending in the out-patient room, but the direct vision early in April was no better than $\frac{2}{60}$ in the right eye and $\frac{1}{60}$ in the left.

She was given iodide of potassium and iron and arsenic at different dates, and on May 1st the fields were recorded as full. On May 22nd she had been freer of headaches, and a month later the vision was, in the right eye $\frac{6}{60}$ and in the left eye nearly $\frac{6}{60}$, but again some headaches and some giddiness three or four times a week had been experienced. At this date I recorded that there was rather an unusual development of hair for her sex and age upon the upper lip, and this, apart from the delayed onset of menstruation, is the only indication of sexual abnormality. In June she was ordered 2 gr. of thyroid extract twice a day and this she took and tolerated well, but later in the year it was supplemented by an acid strychnine mixture.

In October she was of opinion that the vision was decidedly better, and on test types the right eye read $\frac{6}{36}$, the left eye $\frac{6}{60}$. A further X-ray picture of the sella turcica was taken at this date; the fossa was found to be

normal in size and outline, but there was a domed shadow which formed a roof over the sella turcica.

January 29th, 1917.—Seen at St. Thomas's Hospital again.

V.R. $\frac{6}{36}$, 1 letter ; V.L. $\frac{6}{36}$, 1 letter.

Onset of catamenia occurred two months ago, when she was æt. 14 years 8 months, and mother thinks her vision better since, so that she has taken up some branch of munition factory work.

O.D.'s are both pathologically pale, as before ; I think the left a little paler than the right. Each field for white charted by myself I found to be quite full. Sent to X-ray department for further skiagraph of sella turcica.

February 8th, 1917.—The further X-ray picture of the sella turcica shows again the domed shadow, roofing, apparently completely, the fossa.

Case 2.—Mary B—, female, æt. 49 years, was seen in the out-patient department at St. Thomas's Hospital on April 7th, 1916, complaining of severe pain on the top of the head, with general weakness, attributed to influenza, and dating from three weeks earlier. The week before her visit she noticed a mist before the eyes on getting up, and the sight became as bad in two days as it was on the day of her visit. With the right eye she could only count fingers at half a metre, and the left eye had hand movements only. The left pupil was almost, if not quite, inactive to light, and the right showed some slight reaction to light. The pupils were dilated, but acted with convergence. She was made an in-patient, and it appeared from more careful inquiry that the sight first failed in the left eye, and that in a single day it had been reduced to its present condition.

The sight in the right eye began to fail in a similar manner about four days later. There was no family history of defective sight. The diagnosis of retro-bulbar neuritis was first suggested, and an examination by Mr. Howarth of the nose could detect nothing amiss with the accessory air sinuses of the nose.

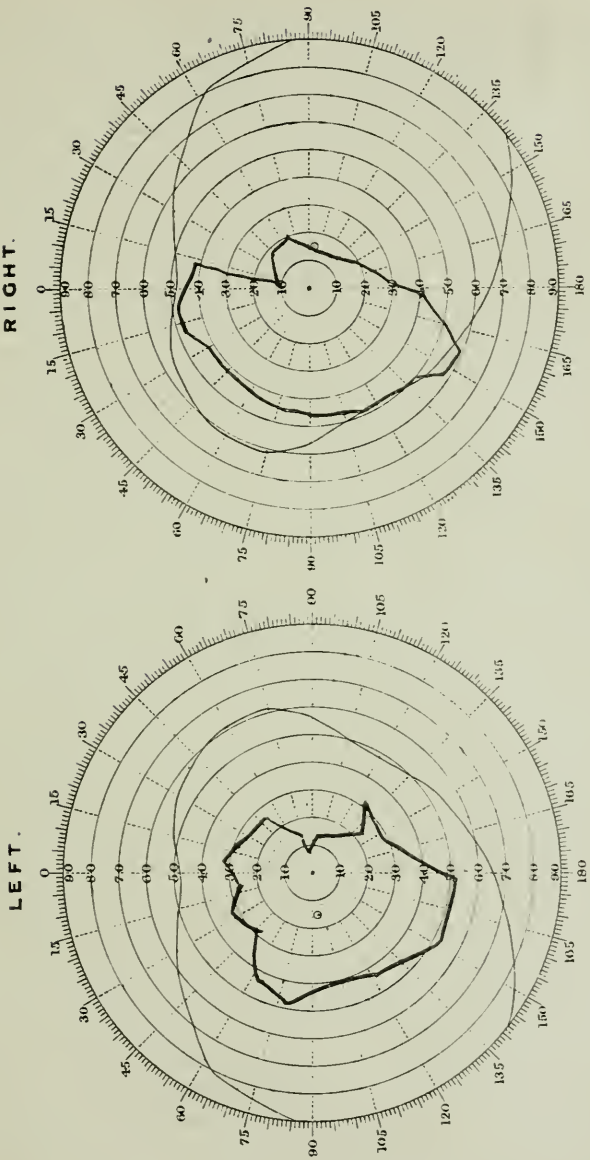


FIG. 27.

The Wassermann reaction was negative. An X-ray examination of the sella turcica was recommended and showed, according to the X-ray officer, enlargement of the pituitary fossa and some want of distinction of the posterior clinoid process, but examination of the plate did not satisfy me that there was any certain departure from the normal.

The patient did not put on weight and was not drowsy.

The thyroid gland was scarcely detected on palpation. The catamenia had ceased when she was æt. 39 years. Fields of vision charted at various specified dates are shown, but cannot be said to be characteristically hemianopic, whether for white or for colour.

On May 3rd a glucose meal of 200 grm. was administered at 4.30 p.m., and Fehling's test showed the presence of glucose in the urine at 8 p.m.

The patient thought that her vision was somewhat variable from time to time. She was examined by Dr. Birley at my request, who considered that there was some weakness of the left face and leg and no obvious change in reflexes, possibly slight drooping of the right upper lid. He stated, in his opinion, the patient had something pressing on the right side, involving, besides the visual fibres, the right third nerve.

Under treatment with thyroid extract, 4 gr. twice a day, and also 10 gr. of anterior lobe pituitary extract twice a day, the patient gradually felt better in herself and obtained relief from headaches. She continued to take pituitary extract and strychnine, and has done so up to the present date, but without any recovery of sight. The fields remain good.

The latest X-ray pictures (January, 1917) show a sella turcica which is probably normal in size, but the shadow shows an approximation of the posterior to the anterior clinoid process which is certainly much greater than is usual, though there is no complete domed roof in this case. The aspect of the picture is, however, decidedly more marked than that presented by the skiagraph taken in April, 1916, some nine months earlier.

Fields charted on February 22nd, 1917, show that no material change has taken place in the preceding ten months. The right field shows a fairly complete temporal loss as before; the left is contracted, but not typically hemianopic. She will now take some thyroid extract for a time.

April, 1917.—There has been no change in the condition of the patient.

Sir George Berry said he took it that Mr. Fisher had been induced to seek to establish the association between abnormality of the sella turcica and Leber's disease—stationary scotomatous atrophy—from the result of the examination of the first cases which he showed before the Society last year, in which the skiagrams showed the sella turcica area occupied by a honeycombed shadow. One of his Edinburgh colleagues, Dr. Traquair, had been recently working at the subject of acromegaly, and had his attention greatly drawn to that region. In talking over the matter with Dr. Traquair, he showed him a number of skiagrams of the sella turcica. Sir George promised that if the matter were brought up at this congress, he would explain the view which Dr. Traquair held as a possible explanation of the appearances which were described by Mr. Fisher. Dr. Traquair believed that the honeycomb or trabecula-like shadow, which at least partially covered the area of the sella turcica, was due to the presence of air cells above the root of the zygoma, such as occurred in some specimens of pneumatic or cellular temporal bone. It appeared that in the formation of the bone there might be an extension of cells in different directions, and in a skiagram taken in the only position which gave a view of the sella turcica, the shadow of these cells would be projected over the region of the sella turcica. A tilting of the patient's head would accentuate it owing to the combined effect of a shadow from both sides. He wished to ask Mr. Fisher particularly whether that source of fallacy could be excluded in his cases. So far as he knew, Dr.

Traquair's skiagrams were all taken in the ordinary way, but with very great care, as he was anxious to locate the sella turcica in connection with his investigations on acromegaly. Could the matter be cleared up by taking stereoscopic pictures? If the suggestion he had just communicated were correct, would not that vitiate the assumption of a connection between the appearances seen in this region and Leber's disease?

Mr. Inglis Pollock, in reply, said the skiagrams in his cases were taken with beautiful sharpness as shown by other parts of the plate; and confidence could, therefore, be placed in the accuracy of the appearances in the sella turcica. Both Dr. Macgregor, who was responsible for the skiagraphy, and he himself considered that the shadow was not due to air cells, and that there must be some abnormality in the pituitary region, as they had never before seen anything like it. He would, if possible, bring up stereoscopic skiagrams next year.

Mr. J. Herbert Fisher, in reply, said the importance of the radiographic picture was very great in settling the question of the possibility of the pituitary body being responsible for the atrophy which was known as Leber's hereditary optic disease. At the same time, in the paper which he read before the Society last year, he brought forward a series of arguments which had led him to expect to find that the pituitary body was responsible. The first case of the disease which came under his observation afforded him an opportunity of getting an investigation of the sella turcica made by an expert radiographer, and then this honeycomb-like shadow was observed. It had not been seen in association with any other condition, though the sella turcica must have been X-rayed many thousands of times. Therefore, the argument in favour of what he advanced by no means rested solely on the presence of a shadow in the sella turcica. The suggestion was, that there was a disorder of the pituitary body, which, in some cases, might be associated with a deposit of calcareous or other salts, which, on the application of

X rays, were capable of casting a shadow. There were many other arguments which he advanced a year ago, leading to the same conclusion. The two cases of which he had just shown pictures, one of which had been watched in process of developing a complete dome-shaped roof over the sella turcica, while the other showed an approximation to that by a drawing together of the anterior and posterior clinoid processes, seemed not to fit in with the idea that a cellular element in the temporal bone projected its shadow into the sella turcica. He was unable to interpret the shadows in the way Sir G. Berry had suggested; he had no knowledge of the best way to show the presence or absence of a shadow in the sella turcica, for that he depended on the expert radiographer. The men who had taken the skiagrams of these cases for him had been experts in their own line, and although they had radiographed many hundreds of sella turcicas, they had not observed these appearances in any other cases than those of bilateral optic atrophy, in some cases with a familial history, in others, one or two, without such history. He would hesitate very much before he agreed that the appearance was due to a shadow cast by the squamous portion of the temporal bone into the region of the pituitary fossa. Whether that could be finally settled by X-ray pictures being taken in any particular way he was unable to say.

XI. DISEASES OF THE LENS.

1. *The history of couching for cataract.*

By R. H. Elliot.

The History of Couching.

The operation of couching for cataract is one of the most ancient procedures known to surgery, the earliest description of the method being that given by Celsus, a contemporary of Christ's. The first historical mention of ophthalmic surgeons was in Alexandria, at the time when medicine and surgery underwent separation from each other in that great and flourishing city, nearly three centuries before the dawn of the Christian era, and Galen states that some of these surgeons devoted themselves exclusively to operating on cataracts. Celsus speaks of the writings of a famous Alexandrian surgeon, named Philoxenes, who lived 270 years before Christ, and from whom he apparently derived much of his lore. These writings have unfortunately been lost, thus yielding to Celsus the proud position of being the first author whose description of the operation has come down to modern times. Sprengel is of the opinion that couching was not only known long before the time of Celsus, but also that the technique of the operation, even at that distant era, varied widely in the hands of its different exponents. Of the correctness of this view there can be little doubt. Sir John Bland-Sutton has recently published a most interesting memoir on the recovery of the sight of Tobit at the hands of his son Tobias, as described in the Apocrypha, and has included in it a copy of Rembrandt's

picture of the famous operation. Whether the displacement of the lens was due to the rubbing employed or to more definite operative measures must be left to speculation, but, in considering this point, it is worth remembering that the Eastern coucher of to-day hides the fact that he is performing an operation under the cloak of the application of a medicinal paste. Nor must we forget that the anointing of the eyes of the blind with clay played a leading part in one at least of the New Testament miracles, and is suggested in a second. It is to be remembered that the Founder of Christianity took His examples from, and moulded His teachings by, the aid of things familiar to the people in their everyday life. The influence of the Oriental on the introduction of couching to the Western surgeon is shown by the repeated references in the history of the subject to Eastern exponents of the procedure. Thus, Razes speaks of the work of an Indian named Tabri, and Avicenna, himself an Arabian, describes at length the instruments and technique of the Arab cataract operators. Abu El Kasim's name proclaims his Arab parentage, despite the fact that he is spoken of as a Spanish surgeon, and the conviction is deepened by the fact that he spoke of the Arabs in Spain as confining themselves to couching in the treatment of cataract, showing he was in intimate touch with them. Nor must we forget to mention the work of Haly Abbas, and of his distinguished son Jesu Haly.

When we endeavour to ascertain the probable date of the first invention of the operation in the East, the fog of uncertainty closes down over us, obliterates all trace of our quest, and drives us to fall back on inference. Those who have spent their lives in an Eastern land know the unbending force of tradition, the hereditary character of occupations, and the intense conservatism of Oriental peoples. All these influences are against change of any kind, and greatly retard the spread of new ideas. When we consider an operation like couching, which is well known over the whole of the East, and which meets in the

simplest manner an age-long need, felt in every village of a tropical or subtropical country, it is not difficult to believe that the procedure may have been one of the early fruits of advancing civilisation, far away back in Babylon the Great, or even earlier still in the home of the Pyramids, tens of centuries before the dawn of the Christian era. From these attractive speculations we must return to weigh the literature of our subject, of which the foundation was so well and truly laid by the great Celsus. His description of the technique he employed is as follows:

"Before the operation the patient must use a spare diet. . . . After this preparation he must sit in a light place, in a seat facing the light, and the physician must sit opposite the patient on a seat a little higher; an assistant behind taking hold of the patient's head, and keeping it immovable, for the sight may be lost for ever by a slight motion. Moreover, the eye itself must be rendered more fixed by laying wool upon the other and tying it on. The operation must be performed on the left eye by the right hand, and on the right by the left hand. Then the needle, sharp-pointed, but by no means too slender, is to be applied and must be thrust in, but in a straight direction, through the two coats, in the middle part betwixt the black of the eye and the external angle opposite to the middle of the cataract. . . . The needle must be turned upon the cataract and gently moved up and down there, and by degrees work the cataract downward below the pupil; when it has passed the pupil, it must be pressed down with a considerable force that it may settle in the inferior part."

Further details follow. To put the matter shortly in modern terminology, Celsus introduced a needle through the sclera and choroid into the vitreous chamber, and depressed the lens from behind, after first rupturing its posterior capsule by vertical strokes made with the point of the instrument. Galen (born A.D. 131) states that there were both in Alexandria and in Rome surgeons who confined themselves to operating upon cataract. Apparently

he also described his own procedure, for some five centuries later Paulus Ægineta (*circa* A.D. 630), in detailing his technique, gave Galen the credit for it. There is practically no difference between the method they both employed and that originally laid down by Celsus. A point of real interest in this connection is that the description of the operation given by Paulus is practically the only one extant from the pen of a Greek author, though not a few of them mention couching and advocate it. For the next landmark in the study of the subject we have to pass over three and a half centuries, till we come to the writings of Avicenna (*circa* A.D. 980), in which we find introduced a new feature in the technique; for he mentions that the Arab surgeons used two instruments for couching—viz., a two-edged lancet with which they made a corneal incision, and a needle with which they depressed the cataract, after introducing it through the incision thus made. In this needle there was an eye near the point through which a thread was inserted. According to Avicenna, the object of this was to help depress the lens, but it seems at least possible that the thread passed through this eye was wound round the instrument, and so served as a stop, similar to that used by the Indian coucher to-day. In any case the description is of great interest, linking as it does the Mohammedan operator of the twentieth century with his predecessor of the tenth. The famous Spanish surgeon Abu El Kasim adopted exactly the same technique for couching as that we have just described. This, as has already been suggested, is not in the least remarkable, for his name bespeaks his Arab descent.

The next description of the operation, which claims our interest, is that by Benvenuto (Benevenutus Hyerosolimitanus), who flourished in the twelfth century. The quaint blending of religion and science, which it reveals, makes it very attractive reading: "Towards the third hour, the patient having fasted, thou shouldst make him sit astride of an ordinary chair, and thou shouldst sit before him in the same way. Keep the good eye of the patient shut,

and begin to operate on the bad eye, in the name of Jesus Christ. With one hand raise the upper lid, and with the other hold the silver needle, and place it in the part where the small angle of the eye is. Perforate the same covering of the eye, turning the instrument round and round between the fingers, till thou hast touched with the point of the needle that putrid water which the Arabs and Saracens called Mesoret, and which we call cataract. Then beginning from the upper part, remove it from the place where it is before the pupil, and make it come down in front, and then hold it for as long as it takes to say four or five paternosters. After, remove the needle gently from the top part. If it happens that the cataract reascends, reduce it towards the lower angle, and when you have introduced the needle into the eye, do not draw it out unless the cataract be situated in the place described above; then gently extract the needle in the same way as you put it in, turning it about between the fingers. The needle being extracted, keep the eye closed and make the patient lie flat on a bed, keeping him in the dark with his eyes shut, so that he does not see the light or move for eight days, during which time put white of egg on twice a day and twice during the night."

Passing over four centuries, we come to an even more interesting description of the operation from the pen of Bartisch of Dresden: "The day being decided upon, on which the operation is to be performed, the doctor who is obliged to, or who wishes to do it, must abstain from wine for two days beforehand. The patient must also fast the same day, and must neither eat much nor little till an hour after the operation. Given the aforesaid conditions, try and procure a well-lighted room, in which the patient may have everything necessary for going to bed and remaining there, as he ought not to be taken to any place far off; the nearer to bed the better. Set thyself on a bench in the light and turn thy back to the window. The patient may be seated on a chair, a stool, or on a box, before thee and near to thee; in any case he is to be

seated lower than thyself. His legs between thine and his hands on thy thighs. A servant stands behind to hold the patient's head. The servant should bend a little, so that the patient may rest his head against him (Fig. 28).

Fig. 28.

The position of the patient, operator, and assistant as depicted by Bartisch.

When the patient is blind of one eye only, the other eye should be bandaged with a cloth and a pad so that he cannot see. Then take the instrument or the needle in one hand, so that the right hand will be for the left eye, and *vice versâ*. With the other hand separate with great care the upper lid from the lower, using the thumb and

the first finger, so that thou canst see how to direct the needle into the eye. When thou wishest to introduce the needle, the eye must be turned towards the light and looking straight at thee; also, I should make the patient

Fig. 29.

To illustrate the method of introducing the needle in Bartisch's operation (Albertotti). The cataract is seen *in situ* in the right eye and couched in the left eye.

turn his eye a little towards his nose, so that thou canst use the instrument better and that thou wilt not injure the small veins of the eye, but respect them. Direct the needle straight and with attention over the membrane called the conjunctiva, straight towards the pupil and

uvea, at the distance of two blades of a knife from the membrane called cornea or from the grey that is in the eye. Hold the needle quite straight, hold it steady so that it will not deviate or slip. Hold the needle and press it, and turn it with the fingers in the eye with great gentleness, according to the instructions you may gather from the figure, which shows an eye in which the cataract has been taken away, while the other eye has not been touched (Fig. 29). Hold the needle firmly while turning it round, and be careful always to have the point towards the middle of the eye, that it almost touches the pupil and the uvea; and not to oscillate by any chance towards one side. When thou feelest that the needle has penetrated into the eye, that it almost touches the pupil and the uvea, and when thou hast proved to be really in the eye, hold the needle securely and move it, letting it slip backwards and forwards towards the pupil till thou art certain of being in the substance of the cataract, which thou canst easily be sure of by the movement of the cataract material. When thou hast remarked that, lower it carefully and gently and slowly, so as not to disturb the cataract; but try and free the matter entirely from the pupil and from the uvea with care, and keep it intact. Press the said matter with the needle under it, with the greatest care, and when thou perceivest that it is altogether free and loose, draw and direct the needle, with the matter behind it, upwards, and then pass it well downwards, behind the thin retina and the aranea of the eye; and take care that it remains there. . . . This is the recognised instruction, research, and indication of the means of operating, of pricking the cataract, or of the manner in which such an operation ought to be initiated and conducted. But no one ought to undertake such an operation unless he has learnt much and seen much, and unless he is fundamentally taught by intelligent doctors. Unless he is so, it is not well to operate. And it is not wise to trust to any of the brotherhood who happen to be dressed in velvet or silk, and who boast of being great

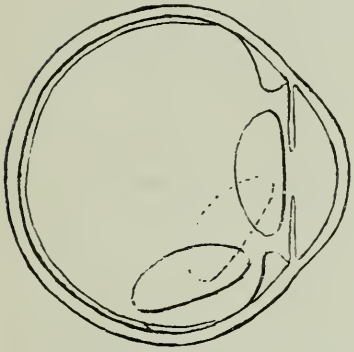
oculists, and are capable of curing the blind from cataract. Certainly these can make holes in the eyes, but I do not know how they can succeed."

The knowledge which the Greeks and the Arabs possessed, before and after the dawn of the Christian era, on the subject of the pathology and treatment of cataract, appears to have been largely forgotten during the Middle Ages. It would seem that both couching and extraction fell into disuse, and that the surgical treatment of cataract was left for centuries in the hands of wandering charlatans, whose ways brought much discredit upon it. Towards the close of the seventeenth century, Pierre Brisseau, a doctor of Tournay, revived the operation, inventing a needle of his own for the purpose. His advocacy of the method aroused bitter controversy, but it was undoubtedly the best operation in the field until the famous French surgeon Daviel performed his first extraction in 1745, and thus sounded the death-knell of a procedure which had held the pride of place in European surgery for over seventeen centuries. It was, however, many years before couching was definitely abandoned in favour of extraction. Indeed, the author has recently had the privilege of discussing this subject with a distinguished surgeon, who can remember the time when depression was still a recognised method of operating in London. It is a great mistake to suppose that Daviel was the first to endeavour to extract a cataract, for both extraction and suction of cataracts have their roots far back in history. Indeed, Antyllus described his method of extraction at the close of the first century of the Christian era, and there are numerous other references to it in early literature. What Daviel did was to adopt a technique which gave a reasonable prospect of success.

The introduction of reclination, as opposed to depression, by Willburg in a Nuremberg thesis, dated 1785, gave a fresh lease of life to couching in its dying struggle with the operation which was destined to supersede it. England, France, Sweden, Germany, and other countries,

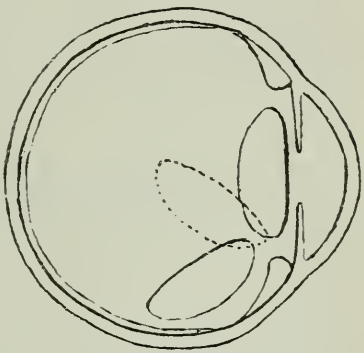
joined vigorously in the discussion, and amongst the powerful advocates of couching were ranked Percival Pott and William Hay of London, Cusson of Montpellier, and Scarpa of Pavia, whilst Benjamin Bell practised both couching and extraction. The admirable treatise by James Ware on cataract (1812) was all but a death-blow for Celsus's operation. The newer procedure was then well in the ascendant, and only needed time to completely strangle its rival. Notwithstanding this, it was left to

Fig. 30.



Depression.

Fig. 31.



Reclination.

The above two figures illustrate the paths taken by the cataracts during the operations (Mackenzie).

Mackenzie, so late as 1854 (fourth edition), to give the most complete and interesting description of couching to be found in literature. He distinguishes sharply between the operations of depression and reclination. In depression, the lens is pushed directly below the level of the pupil, being made to follow the curvature of the eye, to sweep over the corpus ciliare, until it comes to rest on the lower curve of the eyeball, with its anterior surface directed forward and downward (Fig. 30). In reclination, the lens is made to turn over towards the bottom of the vitreous chamber in such a way that what was formerly its anterior surface now comes to look upward, and what was its upper edge is turned to the rear. The whole lens is swung

backward as if on a hinge, composed of the lower fibres of its suspensory ligament, which still remain unbroken (Fig. 31). He divides the operation of couching into four stages, in only the last of which reclination differs from depression. These are: (1) the pushing of a special needle (Fig. 32) through the coats of the eye at a distance of $\frac{1}{6}$ in. behind the temporal edge of the cornea, and to a depth of $\frac{1}{5}$ in.; (2) the laceration of the posterior capsule of the lens by vertical movements of the point of the needle, to prepare an aperture for the passage of the lens; (3) the passing of the needle into the anterior chamber around the edge of the lens, and the laceration of the anterior capsule by vertical strokes; (4*a*) to depress the lens, the point of the needle is carried over its upper

Fig. 32.

Scarpa's reclination needle.

edge, and the handle is raised a little above the horizontal, thereby correspondingly lowering the point, which forces the cataract downward out of sight behind the pupil: the needle is then withdrawn by rotation; (4*b*) to effect reclination, the needle-point is raised not more than $\frac{1}{10}$ in. above the transvere diameter of the lens: its concave surface is pressed against the cataract, which is reclined by moving the handle of the instrument upward and forward, thereby causing its point to pass downward and backward. The cataract is thus made to fall over into the vitreous humour, and is then pressed downward, backward, and a little outward. Mackenzie adds many interesting details as to the modifications of the operation, according to the variety of the cataract to be dealt with, and as to the after-treatment and complications met with.

We come now to a very interesting phase in the study

of the operation of couching. We have shown reason to believe that, like many another valued heritage of the West, it was brought there originally by Wise Men of the East. For more than eighteen centuries it remained a treasured possession of surgery, only to yield its ground before the fierce competition of a method better able to survive the stern test of experience. Slowly but surely its decadence banished it from modern scientific European literature, and then, strangely enough, the advent of Listerism fanned the dying flame of interest in the method; but this time in the East, and not in the West. From the East it had sprung to find a home in the West, and in the East, at the hand of Western surgeons, its last, and by no means least, interesting chapter is in the course of being written. A review of the more recent literature on the subject will establish this contention, and will show how large a share the officers of the Indian Medical Service have taken in the settlement of a question which, apart from its scientific value, has important social and even political bearings.

After a brief visit to India, Hirschberg, in 1894, published an article on couching, in the course of which he spoke favourably of the results of the operation. He was, unfortunately, handicapped by his ignorance of the natives of India and of their ways and customs, with the result that his views on the subject are of comparatively little interest to us. In the following year Capt. H. E. Drake-Brockman described the operation of couching as explained to him by one of its Indian exponents. The latter pierced the sclerotic with a small lancet in the lower outer quadrant close to the cornea, and then introduced a copper needle; "a series of motions of the hand are made from the position on first introduction of the needle to a point corresponding to it in the upper section of the outer diameter of the eyeball." The depression of the lens appears to have taken place next, but the description is throughout somewhat vague. Presumably the operation was the same as that described by Ekambaram, but the

coucher does not seem to have been able to make the steps of the procedure as clear as that surgeon has done.

Henry Power, in the *British Medical Journal* (October, 1901), entered a plea for the occasional performance of the operation of depression in cases of cataract. His experience went far enough back to enable him to remember the time, not only when he had seen surgeons of repute employ this method, but when he had himself imitated the example thus set. His own practice had been to attack the cataract, *viâ* the sclerotic, through the posterior capsule. He framed a number of indications which to his mind justified the occasional performance of couching. It is safe to say that very few of these would be seriously entertained by surgeons to-day. The most interesting point he made was in connection with Himly, in whose work, published in 1843, the statement occurred that "severe inflammation rarely followed reclination, and when it did it often cleared up without leaving any bad consequences." A doubt as to the reliability of Himly's statements is suggested by his claim that he had only two failures in fifty cases, one of these not being attributable to the operation. This is so much at variance with other experience as to make one sceptical about accepting any of his assertions without some reservation.

The next paper of value that we come to is by Maynard (1903). In this he analysed sixty-three cases of couching, which he had met with in Indian practice, and recorded the anatomical examination by Parsons of a couched eye sent home for the purpose. The same year saw the appearance of a paper by Albertotti of Medina, in which that writer somewhat fanatically and unconvincingly advocated a return to couching, with the use of a corneal puncture and with the employment of special instruments for the purpose. A year later he was followed along the same lines by Basso of Genoa, whilst Quartillera published a paper whose recommendations were very similar to those made by Henry Power. In 1905, Major Henry Smith of

Jullundur, in a very outspoken article in the *Indian Medical Gazette*, expressed the opinion "that lens couching at the present time is an operation which should not be practised outside the ranks of charlatans," and added that "it is no easy matter to completely dislocate the lens, and in my observation the partial dislocation is more frequent than the complete in the hands of adepts of the art." In reply to this paper, Maynard reaffirmed his belief that couching is "justifiable under certain conditions." The editor of the *Indian Medical Gazette* invited further discussion of the subject, and in accordance with this request the writer published his statistics based on 125 cases of couching, carefully recorded on printed schedules. In the course of that paper he voiced his strong opposition to the adoption of the Indian operation, or of any modification of it, in the hands of surgeons who enjoy the unique opportunity of obtaining manipulative skill granted to those who work in India. A former pupil of his, Dr. Ekambaram, studied the ways of the Indian coucher at first-hand, and gave the results of his experience in one of the most valuable contributions to the subject yet made. This was in 1910. Two years later the writer was able to review the statistics of 550 consecutive cases of couching, all of which had been carefully noted. Still more cases accumulated before he left India, and by the kindness of Major Kirkpatrick, the total under review has now reached 780, 54 of the eyeballs having been subjected to an anatomical examination.

The examination by J. H. Parsons of a couched eye has already been mentioned. In 1913, A. C. Hudson sectioned and described a similar specimen sent him from India by the writer. The only previous published records of the same kind are from the pen of E. Treacher Collins, and refer to four specimens of couched eyes in the Museum of the Royal London Ophthalmic Hospital. Major H. Kirkpatrick has recently examined several more cases in Madras, and has kindly communicated some of the more interesting of his findings to the writer.

Communications that have been made from time to time before meetings of ophthalmologists show that British surgeons of the first rank are still in favour of performing couching under certain special conditions. A marked instance of this is to be found in the discussion which took place before the Ophthalmological Society of the United Kingdom on February 8th, 1906, following the presentation of a case by Holmes Spicer. On that occasion Rockliffe and Treacher Collins stated that, like Spicer, they had performed the operation in exceptional cases, and Devereux Marshall and G. W. Roll accorded it a modified support under such conditions. The writer has also learnt from personal communications that other leading surgeons have taken a similar line. There for the present we must leave the history of this operation, whose origin is lost in the dim mists of antiquity, and whose chequered career forms one of the most interesting pages in the literature of medicine.

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XII. INTRA-OCULAR TENSION.

1. *The sequelæ of hypotony.*

By E. Treacher Collins.

(With Plates V, VI and VII, figs. 1, 2, 3.)

Increase of intra-ocular tension, hypertony, or "glaucoma" may occur in connection with several different diseases of the eye. We have come, therefore, to regard the sequelæ to which it gives rise as symptoms which may be added to those of several other affections, and which may be considered apart from those of the disease which gives rise to it. Though we speak of one condition as "primary glaucoma," because increase of tension and its manifestation are essential characteristics of the disease, we know that even in it there must be some condition of the eye which precedes and causes the rise of tension.

Owing to the increase of tension being the essential characteristic of primary glaucoma, its sequelæ have collectively and individually received much attention.

The manifestations of the opposite condition, decreased intra-ocular tension, or hypotony, have individually been subjects of careful consideration, but they have not been dealt with collectively in the same way as those of hypertony; in that they, like those of hypertony, occur as something apart from the diseased states which cause them, their consideration as a group will, I think, be found both interesting and instructive.

The sequelæ of hypotony are brought about either

from the relaxation of tissues usually kept taut, or from altered conditions in the circulation. As, however, nearly every part of the eyeball may become affected, it will, I think, be best to consider the alterations in each of its anatomical constituents separately.

A slight degree of minus tension may exist for a considerable time without any apparent detriment to the eye that can be detected clinically, and without any diminution of the visual acuity. Of this we have evidence in cases where a sclero-corneal trephining operation has been peformed for glaucoma, and where subnormal tension has resulted. I have watched such cases with continuous minus tension for years with apparently no ill-effects.

The sequelæ of hypotony are variable, some may be present in one case and absent in another. Though all parts of the eye may be involved, all parts are not always involved to an equal extent. In the normal eye the intra-ocular pressure in the aqueous and vitreous chambers, estimated by the manometer, is found to be the same. Conditions may, however, arise, due to injury or disease, in which the pressure in the one chamber may become lower than that in the other; a change which manifests itself clinically by increase or decrease in the depth of the anterior chamber. The sequelæ of hypotony become most marked in the parts surrounding the chamber in which the tension is lowest.

Cornea.—All those who have had experience of cataract extractions know that the degree of rigidity of the cornea varies considerably in different patients. As a rule, after the completion of the section at the sclero-corneal margin and the escape of the aqueous, it maintains its normal contour. Occasionally, however, it is so flaccid that the atmospheric pressure causes it to fall backwards and become all crumpled on the surface.

A relaxation of the state of tension of the cornea in cases of hypotony tends to throw its two limiting elastic membranes into folds; the extent of the folding varying in amount with the degree of natural rigidity of the tissue.

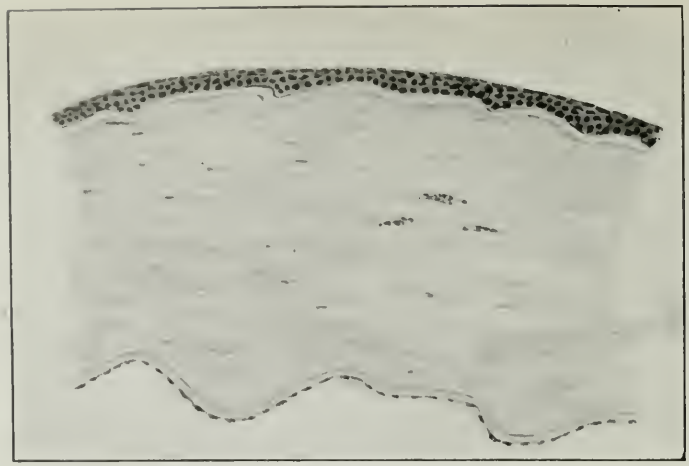

Fig. 1.

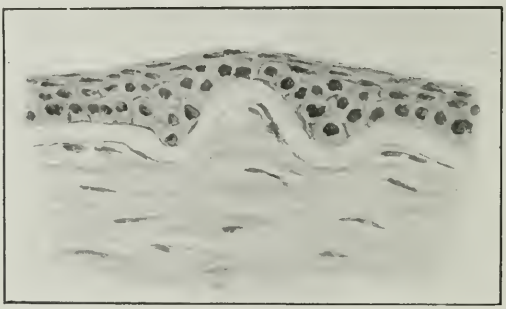

Fig. 2.

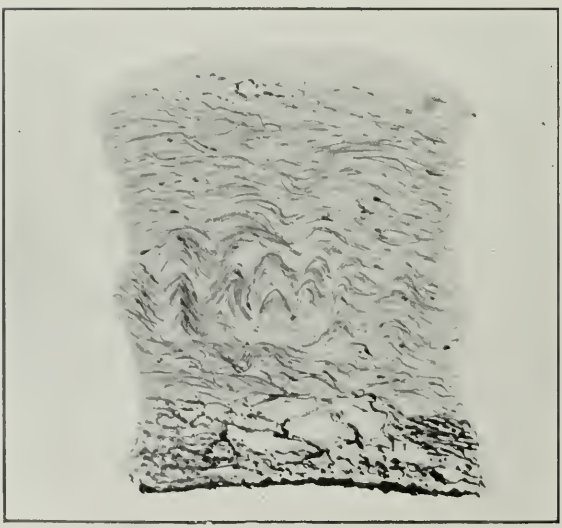

Fig. 3.

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PLATE V.

Illustrates Mr. E. Treacher Collins's paper on The Sequelæ of Hypotony (p. 281).

The figures show the microscopical appearances of the cornea and sclerotic in an eye with reduced tension.

Fig. 1.—A section of the cornea with rucking of both the anterior and posterior elastic laminæ, the excursions of the folds in the latter being much greater than those in the former.

Fig. 2.—Rucking of the anterior elastic lamina shown under higher magnification. Where the lamina projects forwards the covering epithelium is thinned, and where it projects backwards it is thickened.

Fig. 3.—A section of the sclerotic showing wrinkling and spacing out of its fibres, which results in thickening of the membrane.

The examination of a section of a cornea microscopically, in which the wrinkling in both membranes is well marked, shows that the excursions of the convolutions produced in the anterior elastic lamina are less extensive than those in the posterior (Plate V, fig. 1).

The convolutions in the anterior elastic lamina produce projections forwards and backwards, tending to make the surface of the cornea uneven. This unevenness becomes compensated for by the surface epithelium, which endeavours to make good any irregularities by becoming thickened over the depressions and thinned over the elevations (Plate V, fig. 2). Where very marked and abrupt bends forward take place, the thinning of the epithelium is considerable, and sometimes does not suffice to produce a complete restoration in level of the surface, so that ridges are formed.

That the superficial network of grey lines in the cornea described as "reticular opacity" or "lattice-work keratitis," are due to foldings of Bowman's membrane, was suggested by Dimmer (1) in 1890.

Last year Messrs. Holmes Spicer and Affleck Greeves (2) brought forward definite anatomical proof that superficial grey double-contoured lines in the cornea, for the most part running vertically, are due to ridges in Bowman's membrane. Mr. Spicer also showed their association with hypotony. He recorded several cases, of what has been previously described as "ophthalmomalacia" or "essential phthisis," in which these lines were present. Commenting on his series of cases, he says: "One very remarkable feature which is almost always found during at least some period of the attack is the softening of the eye, so that the tension falls to − 1 and often to − 2. During this time of diminished tension the vision is greatly impaired, and in one case it dropped to $\frac{2}{60}$. The attack itself lasts from a few days to a week or longer, and, as the ridge disappears, the vision recovers, and the tension becomes normal again."

Further, in commenting on the nature of the cases, he

says : " It is most probable that diminished tension is the essential feature, and that the lines are produced by a kind of crumbling or folding of the relaxed Bowman's membrane. This view of it was first suggested by the behaviour of the earlier cases that I saw, in which an injury to the cornea treated by a compress was followed by the appearance of these lines. These seemed to be the result of pressure, as they disappeared soon after the use of the compress was discontinued."

In his pathological examination of these lines in the cornea, Mr. Greeves found that beneath the wrinkled Bowman's membrane there was some new formation of fibrous tissue which, as he says, could only have been produced by the activity of the corneal corpuscles in the substantia propria, since in no case was any vascularisation of the tissue found. This increased activity of the corneal corpuscles may, I suggest, best be accounted for by lymph stasis due to the kink in the membrane. It was clearly not the result of true inflammation.

The central part of the cornea is the place where rucking of the anterior elastic lamina is most likely to occur as the result of hypotony. The superficial grey lines, in " reticular opacity " and in the series of cases described by Mr. Spicer, have never been found to extend to the margin of the cornea. A peculiar feature of the lines noted by Mr. Spicer is the appearance in them in places of nodules of greater density, and sometimes of greater thickness. These may be due to irregularities in the folds of the membrane, or possibly to the breaks in the continuity of the membrane where the nerve fibres perforate it to come forward to the epithelium.

The rucking of the posterior elastic lamina which results from relaxation of tension may produce striæ of opacity situated deep in the cornea. Hess (3) and Schirmer (4) have described the striæ of opacity which extend downwards from a wound after cataract extraction as due to folding of the posterior layers of the cornea, including Descemet's membrane, consequent upon

the section. Striæ of a similar character are met with sometimes in connection with hypotony in an intact eye, and after the operation of sclero-corneal trephining where the tension has for a time remained low.

Fuchs (5), describing this form of opacity in his text-book, says: "A similar striate opacity of the cornea is sometimes observed in cases of detachment of the retina, which have been treated with the pressure bandage. The eye becomes suddenly very soft and the anterior chamber remarkably deep, and in the cornea fine grey striæ show themselves, which cross in different directions, so that the opacity looks like creased tissue paper. Here also, without doubt, folds in the cornea take part in producing this effect (Deutchmann, Nuel)."

Microscopical examination of sections of corneæ, with convolutions of the posterior elastic lamina from hypotony, show that the posterior layers of the substantia propria take part in the rucking, and follow the bend of the convolution of Descemet's membrane. Increase of intra-ocular tension by stretching the cornea, tends to the approximation of the layer of fibrous tissue of which it is composed, and compression of the spaces for the circulation of lymph between them. As a consequence of glaucoma we find the cornea becomes hazy, anæsthetic, and badly nourished.

Decrease of intra-ocular tension by relaxing the tissue of the cornea favours a widening-out of its lymph spaces, and an increase in the supply of its circulating nutritional fluid. It is the increased supply of nutritional fluid which follows the diminution of tension produced by a Sæmisch's section, or paracentesis, which accounts for the beneficial effects of these operations in ulceration of the cornea. To obtain the maximum benefit from a Sæmisch's section it is directed that the section must be reopened daily; *i. e.* the state of hypotony must be maintained. From experiments with Schiöt's tonometer we know that eserine applied to the eye is capable of decreasing normal tension. It would, therefore, tend to increase the lymph flow through

the cornea, and the benefit which some surgeons have found from its use in cases of ulceration of that membrane, may thus be accounted for.

Sclerotic.—In the normal state of distension of the globe the bundles of fibres of which the sclerotic is composed run a straight course, and are kept compressed in contact with one another. When the tension of the eye becomes reduced they assume a wavy outline and spaces open out between them. The effect of this alteration in the course of its fibres is to cause a general thickening of the membrane (Plate V, fig. 3).

The rigidity of the sclerotic in the normal state is such, that when the eye has been removed from the body, and had the contents evacuated, it will still retain approximately its usual curvature. When, however, the tension of the eye becomes markedly decreased, the pressure exerted upon it by the surrounding muscles causes the sclerotic to become puckered. This puckering first takes place immediately behind the insertion of the recti muscles. At the site of their insertion, the prolongations of Tenon's capsule, which extend from their external surface to the walls of the orbit, the so-called "check ligaments," hold the sclerotic in the anterior part of the eye on the stretch and prevent it falling in. The oblique muscles, which encircle the globe behind the insection of the recti muscles press on its sides, and when the sclerotic is not kept distended by the intra-ocular contents, cause it to collapse inwards. If diminished tension persists, in course of time, the anterior part of the globe which is kept stretched out by the check ligaments, becomes drawn back, by the pull of the recti muscles, over the collapsed posterior part, so that the depressions formed beneath the recti muscles gradually tend to increase in depth. Sometimes the posterior part of the sclerotic becomes so crumpled that two contiguous folds are formed in it (Plate VII, fig. 1).

Choroid.—A comparison of sections of eyes, in which previous to removal the tension was increased, with others,

Fig. 1.

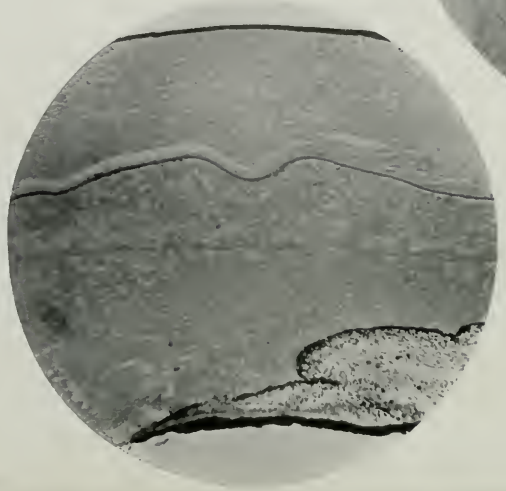

Fig. 2.

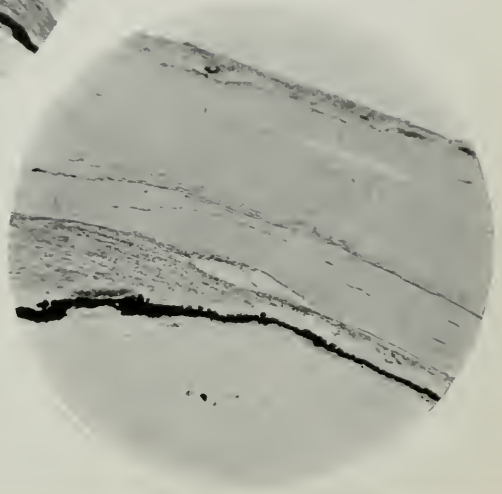

Fig. 3.

PLATE VI.

Illustrates Mr. E. Treacher Collins's paper on The Sequelæ of Hypotony (p. 281).

The figures are photographs of sections of an eye, fixed in Zenker's fluid, which contained a coagulum (presenting precisely similar characteristics) between the ciliary body and sclerotic from which it was detached, and filling the anterior chamber. There was no coagulum in the vicinity of the ciliary processes or behind the iris.

Fig. 1.—Shows the anterior part of the ciliary body and part of the anterior chamber.

Fig. 2.—Shows the central portion of the anterior chamber and the pupillary margin of the iris on one side.

Fig. 3.—Shows the posterior part of the ciliary body detached from the sclerotic.

For the photographs from which these figures were reproduced the writer is indebted to Mrs. Elliot.

in which it was decreased, shows a marked difference in the appearances of the choroid. In the former it is compressed, its vascular layers being narrowed and the lymph spaces between it and the sclerotic obliterated. In the latter it is thickened, its blood-vessels being distended, and the lymph spaces, especially those anteriorly, widened out and filled with fluid, giving a granular deposit with Zenker's fixation.

The constant association of distension of the lymph spaces between choroid and sclerotic, so-called "serous detachment of choroid and ciliary body," with hypotony was pointed out by Mr. A. C. Hudson (6). In a consecutive series of 574 pathological-anatomical examinations he met with 153 cases of detachment of choroid and ciliary body, of varying extent, by fluid yielding a granular coagulum on fixation. The conclusions he arrived at from a careful analysis of these cases are as follows :

"(1) That serous detachment of the choroid and ciliary body is the natural accompaniment of considerable reduction of the intra-ocular pressure, and that its occurrence is the rule in every case of sustained reduction of pressure.

"(2) That the degree of the detachment varies, more or less directly, as the degree of reduction of the intra-ocular pressure.

"(3) That the fluid responsible for the detachment is derived probably from the choroidal blood-vessels and not improbably form the veins.

"(4) That reduction in intra-ocular pressure is the chief factor in the causation of detachment of the choroid and ciliary body in shrunken eyes."

Detachments of this sort occur first, and become most marked, in the region of the ciliary body, the part of the eye least accessible to ophthalmoscopic examination. It is not surprising, therefore, that they frequently escape detection clinically. Sometimes a detachment of the ciliary body and choroid occurring after an intra-ocular operation is sufficiently extensive to be seen ophthalmoscopically. Fuchs (7), who looked for it systematically,

found it present in 4 per cent. of his cases of extraction of cataract. He commented on the frequency of its occurrence where, after a successful operation, the anterior chamber is found one morning abolished and the tension reduced. It is more frequently seen after operations for glaucoma, especially those operations which aim at the production of a filtration scar, and in which subnormal tension is likely to be more prolonged than after simple iridectomy. Fuchs found it in 10 per cent. of glaucoma iridectomies, and Meller (8) in more than 22 per cent. of Lagrange sclero-iridectomies. Probably all of us who have done many Elliot's sclero-corneal trephining operations are familiar with its occurrence after that procedure. I have met with it several times, persisting usually while the tension remains minus, and disappearing when it again becomes normal.

Barkan (9) estimates that post-operative detachment of the choroid is to be found in 4 per cent. of cataract operations, 10 per cent. of trephining, and 20 per cent. of Lagrange.

Fuchs attributed post-operative detachment of the choroid to a rupture of the ligamentum pectinatum, due to the operation allowing the aqueous humour to find its way into the supra-choroidal lymph spaces. Such a hypothesis leaves unexplained the accompanying hypotony, as there would be no diminution in the intra-ocular contents. Nor can it be employed to explain cases of choroidal detachment which occur in connection with hypotony where no operation has been performed.

Meller suggested that the fluid causing detachment of the ciliary body proceeded from the blood-vessels of the ciliary processes. The secretion from which, into the interior of the eye, he thought, would not be sufficiently rapid under the altered conditions produced by hypotony.

If this view was correct the fluid poured out from the blood-vessels of the ciliary processes, to reach the supra-choroidal lymph spaces, would have to pass through the ciliary muscle. It is hardly likely to do this without

producing some œdematous changes in that structure. In microscopical sections of eyes with detachment of the choroid no such œdematous changes in the ciliary muscle are to be seen. Hudson's suggestion as to the causation of the detachment is probably the correct one. He regards it, as already stated, to be essentially a sequela of hypotony and due to inundation of the supra-choroidal lymph spaces with fluid transuded from the thin-walled choroidal veins.

Under normal conditions the blood-pressure in the emissary ocular veins fluctuates. Of this we have evidence in the pulsation of the central retinal vein on the disc, due to slight heightening of the intra-ocular pressure caused by increased arterial tension at each systole of the heart.

If the blood-pressure in these veins were always lower than the intra-ocular pressure they would become closed, as they do when the tension is slightly increased by finger pressure. That it is not much higher than the intra-ocular pressure is shown by the slight amount of finger pressure which causes them to collapse. What applies to the emissary veins of the retina, applies also to those of the choroid. Prof. Leonard Hill (10) opened the vortex veins of a cat, allowing the blood to escape, and then forced Ringers' solution into the aqueous. He found that when he raised the aqueous pressure to the blood-pressure the outflow of blood from the veins stopped.

From the experiments of Asayama (11) in rabbits, it would appear that the veins at the anterior part of the choroid and the vortex veins, under normal conditions, act as excretory channels for some of the contents of the anterior chamber and lymph in the anterior portions of uveal tract. He found that some of the particles of Indian ink, when injected into the anterior chamber, passed through the crypts at the base of the iris, gained entrance to the veins of the iris, and by them to the vortex veins.

A decrease of the tension of the eye entirely alters the

normal relation between blood-pressure and intra-ocular pressure. The pressure of blood in the intra-ocular veins is then much higher than the intra-ocular pressure, and instead of the veins in the anterior part of the uveal tract acting as excretory lymph channels, serous fluid transudes through their walls, which, accumulating in the supra-choroidal lymph spaces, causes detachment of the ciliary body and choroid.

A slackening in the tension to which the choroid is normally exposed as a result of diminution in the intra-ocular tension, may cause its elastic lamina to become kinked, and in extreme cases of shrinking of the globe to become much folded and convoluted.

The details of the changes to which the wrinkling of the elastic lamina of the choroid gives rise, have, so far as I know, not been previously described. The following description of their microscopical appearances has been made from an examination of them in sections of a series of shrinking eyeballs. The ophthalmoscopic appearances can at present only be a matter of conjecture.

The microscopical examination of sections of eyes in which the elastic lamina of the choroid was relaxed from its normal state of tension show it thrown into folds, which present large rounded undulations inwards, and abrupt angular projections outwards (Plate VII, fig. 2). The height of the undulations, and the depth of the recesses between them, varies with the degree of slackness of the membrane. When they are shallow there is but little disturbance of the lining pigment epithelium, only slight thickening opposite the angular recesses. When, however, these angular projections externally, become deep, the elastic membrane forming the two sides of the angle becomes so closely approximated that the lining pigment epithelium is unable to extend into the extreme recesses of the fold, at any rate not as two distinct layers of cells. The result is, that some of the pigment epithelial cells become collected together in masses and heaped up internal to the

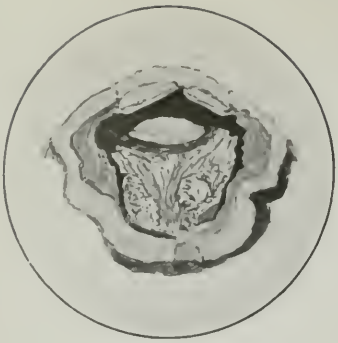

Fig. 1.

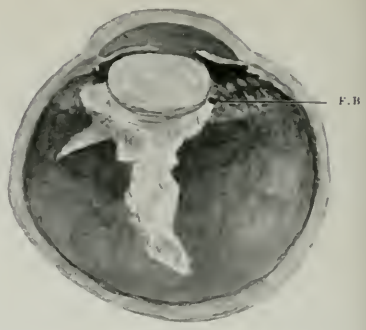

Fig. 4.

Fig. 2.

Fig. 3.

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PLATE VII.

Figs. 1, 2, and 3 illustrate Mr. E. Treacher Collins's paper on The Sequelæ of Hypotony (p. 281).

Fig. 1.—Shows the lateral half of an eye which had diminished tension. The ciliary body and choroid are detached from the sclerotic, and there are indentations in the posterior half of the sclerotic behind the insertions of the recti muscles.

Fig. 2.—Shows rucking of the elastic lamina of the choroid in an eye with diminished tension.

Fig. 3.—Shows more extensive rucking of the elastic lamina of the choroid, under higher magnification, with considerable heaping up of the pigment epithelium on its inner surface.

Fig. 4 illustrates Mr. Arnold Lawson's paper on A Case of Detachment of the Retina at the Ora Serrata (p. 231).

F.B. points to the foreign body and also to the retina at the ora serrata, where it is torn away from the ciliary body. On the opposite side of the specimen, at the point of entrance of the foreign body, a piece of retina is shown still adherent to the choroid.

choroid, overlying the site of the kink (Plate VII, fig. 3). The masses of heaped up cells are sometimes densely pigmented, and if it were possible to view them clinically with the ophthalmoscope, they would probably appear as pigmented streaks or dots beneath the level of the retinal vessels.

Iris.—In the iris, as in other portions of the uveal tract, the effect of hypotony is to produce vascular congestion. This can be observed, not only in pathological specimens, but also clinically. In an albino rabbit enlargement of the vessels of its iris is seen to follow evacuation of the aqueous humour by paracentesis. A similar enlargement of the abnormal vessels, met with sometimes as a sequela of iritis, is seen when the aqueous escapes preliminary to the performance of an iridectomy. In some of these cases, not only are the abnormal vessels seen to dilate, but blood may be observed to ooze from them.

The aqueous humour as the result of hypotony becomes considerably altered in character. Normally it contains only a small percentage of proteids and does not undergo spontaneous coagulation. If the tension of the eye is diminished by a paracentesis of the anterior chamber, the aqueous which is formed while the tension remains low, contains a considerable amount of proteids and will coagulate spontaneously. The increased amount of proteids has been found in the rabbit to persist for six to eight hours. By that time the normal tension has become re-established, and the aqueous regains its normal characteristics.

We have seen that the altered relation of the blood-pressure in the choroidal vessels to the intra-ocular pressure, which occurs in hypotony, results in transudation of serum from the veins on the outer surface of the choroid into the lymph spaces between sclerotic and choroid. Does a similar alteration in the relation of blood-pressure in the vessels of the iris to the intra-ocular pressure result in transudation of serum from the veins on its outer surface into the anterior chamber, and account for the altered character of the aqueous humour?

The fluid transuded into the supra choroidal lymph spaces as the result of hypotony, and that poured out into the anterior chamber under similar conditions, have precisely similar characteristics. Both, with Zenker fixation, give rise to a granular deposit and some fibrinous coagula. I have sections of an eye, in which Zenker's fixation was employed, where this abnormal fluid was found situated both in the anterior chamber and between the choroid and sclerotic. The appearances that its coagulum presents under the microscope is precisely the same in the two situations. Moreover, though the anterior chamber is completely filled with the granular fibrinous coagula none is formed in the posterior chamber or circumlental space (Plate VI, figs. 1, 2, and 3). If in this case, the aqueous had come entirely from the ciliary body, we should have expected it to have yielded the same granular deposition in its vicinity as in the anterior chamber.

The experiments of Nuel (12) and Asayama of injecting Indian ink into the interior of the eye, seem to show very definitely, that under normal circumstances the veins of the iris assist in the excretion of the aqueous humour from the eye. The grains of Indian ink could be traced passing through the crypts in the anterior surface of the iris, becoming aggregated around the veins and capillaries in its middle layers, and passing even into the vessels themselves.

In Nuel's experiments injections were made into the vitreous. Where injections have been made into the aqueous the success of the experiments is said to depend on "Care being taken not to lose aqueous during the injection, as the fresh aqueous secreted may clot and imprison the particles of ink in the meshes of the fibrin network."

Although under normal conditions experimental evidence goes to show that the iris extracts fluid from the anterior chamber and does not secrete fluid into it, under the condition of abnormal hyperæmia which results from inflammation, an exudation from its blood-vessels into the

aqueous undoubtedly takes place, causing it to become turbid and giving rise to the altered colour and dull appearance of the iris.

Eyes are also met with where pathological examination shows that the only possible source for the fluid found in the anterior chamber would seem to be the vessels of the iris. These are eyes in which there is total posterior synechiæ, or where fibrous tissue fills the whole circumlental space and stretches across behind the lens in the ciliary region, the outcome of plastic exudative cyclitis. Cases often described clinically as "pseudo-glioma," in which the anterior chamber at its periphery is abnormally deep, due to retraction of the periphery of the iris caused by shrinking of fibrous tissue adherent to it in the circumlental space, and in which the tension is below normal. The fluid in the anterior chamber cannot have come from the ciliary body, the fibrous tissue filling the circumlental space rendering that impossible, and it is difficult to believe that it could be stagnant fluid remaining in the anterior chamber from some time previous to the cyclitis.

Ciliary body.—A reduction in the tension of the eye, such as that produced by the escape of the aqueous humour through a wound of the cornea, reduces the intraocular pressure in the posterior chamber to that of the atmosphere, and so alters profoundly its normal relation to the blood-pressure in the capillary vessels of the ciliary processes.

It has been found that if fluorescine solution be injected subcutaneously, and the aqueous humour evacuated, green fluid is seen to pass forwards into the anterior chamber through the pupil. It may be inferred, that the increased difference which is produced between the pressure in the capillaries of the ciliary arteries, and in the posterior chamber, has increased the rate of flow of fluid from the ciliary body.

Greef (13) examined microscopically the eyes of rabbits which had been removed at various intervals after puncture of the cornea and evacuation of the aqueous humour.

He found that shortly after the puncture the ciliary processes became extremely hyperæmic, and that sometimes there was diapedesis or small hæmorrhages into their stroma. Also that the epithelium covering the processes became, in several cases, raised up in the form of vesicles, or blisters, which contained a highly albuminous exudate. These vesicles gradually increased in size and ultimately burst, discharging their contents into the posterior chamber. Greef suggested that the large increase of proteids found in the newly-formed aqueous humour was derived from the contents of these ruptured vesicles. It was, however, found by Bauer (14), that there is no parallelism between the time of onset and the disappearance of these vesicles, and the change in the aqueous. Aqueous humour with increased proteids has formed when the vesicles are commencing to develop, and it has regained its normal characteristics long before the vesicles have all disappeared.

E. E. Henderson and Janet Lane-Claypon (15) punctured the cornea in albino rabbits' eyes with a cannula, through which they allowed the aqueous to flow for a varying time from twenty minutes to an hour. They found, as Greef had done, that bladder-like spaces in the ciliary epithelium filled with albuminous material were produced, and agree with Bauer that this appearance is most noticeable when the pressure is rapidly reduced. In slow drainage experiments they were able almost entirely to eliminate them. They also noticed changes in the protoplasm of the epithelial cells themselves of a disintegrating character, which became more marked the longer the drainage continued. It could be detected after as short a period as twenty minutes, and at the end of an hour had gone on to such an extent, that the cells were only represented by a few scattered nuclei and scarcely any protoplasm. Together with this disintegrating process in the epithelial cells, a clot formed on the posterior surface of the ciliary body, which was so intimately connected with it that it was impossible to remove

it completely without injuring that structure. The longer the eye was allowed to drain the denser did this clot become.

These interesting changes in the ciliary epithelium, occurring as the result of prolonged drainage of the aqueous from the anterior chamber, afford no definite evidence as to the source of the abnormal aqueous which is formed ten minutes after paracentesis, and which, as previously stated, there is some reason to think may be an exudate from the veins of the iris.

The observations of Greef seem to show that the sudden reduction of pressure in the posterior chamber, produced by the evacuation of the aqueous humour, causes the blood-vessels of the ciliary processes to dilate and some extravasation of serum through their walls to take place. That this serous exudate is prevented from passing into the posterior chamber due to the resistance offered by the epithelium lining the processes. A resistance which is so great, that the epithelium actually becomes raised up in the form of a vesicle by the exudate, instead of being permeated by it. When one of the vesicles has become greatly distended, its inner wall ruptures, and fluid of an albuminous nature passes into the posterior chamber.

The epithelium of the ciliary processes in rabbits' eyes is apparently not an inert structure, permitting lymph, exuded from the capillary vessels of the ciliary body, freely to filter through it. It seems to exert a selective action keeping back proteids, the presence of which is one of the chief differences between the normal aqueous humour and lymph.

Detachments of the unpigmented layer of cells of the pars ciliaris retinæ from the pigment layer of the ciliary body have frequently been met with in disease conditions in the human eye, an albuminous fluid being found present in the cysts so formed. Such detachments have been described by Kuhnt (16) and Brailey (17), but the association of them, as in rabbits' eyes, with a reduction of intra-ocular pressure due to escape of the aqueous humour has not been noted.

John Griffith (18), who examined the ciliary region of human eyes which had been excised within a short time after perforating injuries, described its epithelial cells as appearing distorted and vacuolated.

Retina.—Diminution of tension is a common accompaniment of "simple" detachment of the retina. Leber estimated it as present in 90 per cent. of such cases. It appears to be mainly due to a diminution of pressure in the vitreous chamber, because the anterior chamber is found to be increased in depth. Fuchs, in his text-book, says: "The intra-ocular tension in detachment of the retina is generally reduced, because the volume of the vitreous is diminished; for the same reason the anterior chamber is often found to be deepened because the lens has sunk backwards."

Is this hypotony a sequela of the retinal detachment, or, is the retinal detachment a sequela of the hypotony?

For the following reasons it seems highly probable that decreased pressure in the vitreous chamber may be one factor in the causation of serous retinal detachments.

(*a*) Injuries of the eyes which we know suddenly reduce the pressure in the vitreous chamber are exceedingly liable to give rise to detachment of the retina, *e. g.* perforating wounds involving escape of the vitreous humour; indirect ruptures of the sclerotic, in which the suspensory ligament of the lens is torn through, and where the vitreous humour comes forward into the anterior chamber or prolapses through the rupture; contusions of the eye without perforation, in which the suspensory ligament is ruptured and the vitreous protrudes forwards into the circumlental space. In such cases the hypotony evidently precedes the detachment.

(*b*) The cases in which simple serous detachments most frequently occur are cases of myopia, in which changes have taken place in the vitreous humour which would be likely to lead to decrease of pressure in the vitreous

chamber, such as liquefaction of the vitreous, or shrinking of it and detachment of the hyaloid membrane from the retina.

(*c*) Decrease of the pressure in the vitreous chamber alters considerably its normal relation to the blood-pressure in the capillaries of the choroidal vessels, the blood-pressure in them then being much higher than the chamber pressure, a serous exudation from the capillaries would be likely to occur. The fluid found beneath the retina in cases of simple detachment is of a serous character, and the most likely source from which it is derived is the capillaries of the choroid, as they normally supply the nutrient fluid which nourishes the outer layers of the retina.

(*d*) Conditions which cause obstruction to the flow of blood in the choroidal veins, thereby augmenting the pressure in their radicles, give rise to serous detachment of the retina. These conditions are: (1) Sarcoma of the choroid, in which detachment of the retina, apart from the elevation of it caused by the new growth, is frequently met with. (2) Albuminuria, in which venous obstruction is general, and where the detachment is usually bilateral and often-accompanied by general œdema. In these cases, when the patient recovers from the general condition, re-attachment of the retina not uncommonly takes place. (3) Orbital cellulitis, in which the obstruction to the choroidal veins is most likely due to thrombosis. In these cases it is an increase in the blood-pressure in the capillaries of the choroid, above the normal pressure in the vitreous chamber that causes the serous exudation, which, passing through the pigment epithelial layer, accumulates beneath the retina. In cases of hypotony there is a decrease of pressure in the vitreous chamber, which in a similar way alters the balance between blood-pressure and chamber-pressure.

Optic nerve.—At this Society in 1901 I showed, together with Dr. Hinnell, a boy with minus tension in one eye, which had lasted for nine months after contusion

of the eye from a shot from an air-gun. In this case the optic disc presented the appearances of choked disc of a high degree. The amount of swelling was estimated to be = 7 D. The retinal blood vessels in the vicinity of the disc were in part obscured. The veins were large and the arteries small. These changes in the disc remained the same during the three months the patient was under my observation, during the whole of which time the tension was − 2. Last year in the paper I read before this Society on "Contusion Hypotony," I described a somewhat similar case. It was that of a boy, æt. 6 years, who was a patient of Mr Eason's. He had a cystoid condition at the sclero-corneal margin, apparently the result of a partial indirect rupture of the sclerotic from a blow against a lamp-post, two and a half years previous to my seeing him. The tension of the eye was − 2 and there were the appearances of choked disc, the height of the swelling being estimated as equal to 5 D.

There was nothing in either of these two cases to account for the swelling of the optic disc apart from the local condition of the eye. Both remained under observation for some considerable time. In both there was a high degree of hypotony of long standing and marked œdema of the optic papilla. Are the two conditions in any way associated?

Interest is lent to this subject by a paper published last year by Dr. Walter R. Parker (20) entitled "The relation of choked disc to the tension of the eyeball, an experimental study." His experiments were made on dogs and monkeys, in which he lowered the tension of the eye by performing a sclero-corneal trephining and increased the intra-cranial pressure in various different ways. One of the conclusions which he arrived at was that, "when the intra-cranial pressure is increased by artificial tumours placed in the occipito-parietal region, the nerve in the eye of least tension is the first to show the choked disc." That is to say, the condition of hypotony favours the development of an œdema of the head of the optic nerve.

Can hypotony by itself be the cause of such œdema, as my two cases seem to suggest?

Experimental evidence goes to show that the central retinal vein serves as an excretory channel for a certain amount of fluid from the vitreous, and probably also for some of the lymph from the retinal tissue. In the same way as the vortex vein, of the choroid serve as excretory channels for a certain amount of the aqueous and of lymph from the uveal tract. It has already been shown that hypotony, by altering the normal relation of the intra-ocular pressure to the blood-pressure in the veins of the uveal tract, results in a condition of œdema of the lymph spaces superficial to the choroid. Not only is the normal excretory function of these veins arrested, but there is an abnormal exudation of lymph through their walls. In a similar way the condition of hypotony, by establishing a much higher pressure in the emissary veins of the retina than in the vitreous chamber, would interfere with their excretory function and tend to produce an exudation through their walls. This exudation taking place chiefly around the larger vessels would result in the appearance of choked disc.

Lens.—In several cases in which I have performed the operation of sclero-corneal trephining for glaucoma I have found, that when I have tested the patient's vision, two or three weeks after the operation, and while the tension is still below normal, that the refraction of the eye has become somewhat increased to what it was previous to the operation. That is to say, if the patient was previous to operation hypermetropic, the degree of hypermetropia is lessened, if emmetropic slight myopia has developed. When I have tested the vision again later on, after normal tension has become restored, I have found that the state of refraction which was present previous to operation has become re-established.

On speaking of this matter to Col. Elliot I found he was quite familiar with the condition, and had observed higher degrees of fluctuation in refraction than I have

done. In none of my cases did the alteration exceed one diopter.

In a case of ophthalmomalacia I have also observed the production of 1 D. of myopia during the lowered intra-ocular tension which disappeared on the return of normal tension.

The most probable explanation of this increase of refraction occurring in connection with hypotony is, that in these cases the lens become slightly displaced forwards owing to the reduction of pressure in the aqueous chamber being greater than in the vitreous chamber. When normal tension is restored and the pressure in the two chambers again becomes equal the lens returns to its normal position.

Another possible factor in connection with this matter is, that a diminution of the tension of the eye may cause relaxation of the suspensory ligament and so allow of an increase in convexity of the lens.

To sum up, the conclusions which my investigations seem to suggest, are as follows :

(1) That hypotony may occur in connection with several different affections of the eye, and that the sequelæ to which it gives rise may be considered apart from the affection causing it.

(2) That the sequelæ of hypotony vary in different cases according to the chamber (aqueous or vitreous) in which the pressure is most reduced, and, according to its degree, duration, and rapidity of onset.

(3) That the sequelæ of hypotony may be classified as follows :

(1) Those due to relaxation of tissues usually kept taut.

(a) Wrinkling of the anterior elastic lamina of the cornea ; producing superficial double contoured lines.

(b) Wrinkling of the posterior elastic lamina of the cornea and layers of the substantia propria immediately adjacent to it ; producing deeply seated striated opacities.

(*c*) Thickening of the sclerotic and the production of puckers in it behind the insertions of the recti muscles.

(*d*) Wrinkling of the elastic lamina of the choroid, and heaping up of the pigment epithelium on its inner surface where the folds are deep.

(*e*) Displacement forwards of the lens and slackening of its suspensory ligament ; both of which tend to increase the refraction of the eye.

(2) Those due to production of relatively higher blood-pressure, compared to intra-ocular pressure, than exists under normal conditions.

(*a*) In the choroidal veins the normal excretion of lymph through them is arrested, and instead an exudation of lymph takes place, giving rise to detachment of ciliary body and choroid.

(*b*) In the veins of the iris the normal excretion of aqueous humour through them is arrested, and instead an exudation of lymph into the anterior chamber takes place ; this exudation alters the normal consistency of the aqueous by increasing its percentage of proteids and rendering it spontaneously coagulable.

(*c*) In the capillary vessels of the ciliary body the secreting function of that structure is quickened, and an exudation of lymph takes place, the albuminous constituents of which, in a rabbit's eye, being unable to permeate the epithelial lining of the part raise it up in the form of vesicles.

(*d*) In the capillary vessels of the choroid the normal exudation of lymph is stimulated, which, if it permeates the pigment epithelium, may collect beneath the retina causing detachment of that membrane.

(*e*) In the retinal veins of the optic disc the normal excretion of fluid from the vitreous is arrested, and an exudation of lymph into the head of the optic nerve takes place, giving rise to the appearances of choked disc.

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Sir Anderson Critchett, said it was not his intention

to debate the President's contribution, because it was one which required so much thought and consideration that one hesitated to speak on it on the spur of the moment. The members present had already indicated the intense pleasure and satisfaction which the paper had afforded them, but he would also like to give them an opportunity of saying how cordially they all welcomed the President to the Chair, and how earnestly they hoped that his period of office would be a very happy and a very successful one. The choice of subject of Mr. Collins' address, the address of one of the greatest pathologists of our time, had been a very happy one. The paper was exhaustive, but by no means exhausting, for it was pregnant with interest from start to finish. And, if he might be allowed to say so here, one of the changes which, he thought, the war had brought upon them was the realisation that we ourselves possessed an innate power in many directions which, for some occult reasons, had never yet been used to its fullest extent. We had allowed foreigners to take our dyes, to take our metals, and to work them very much to our own disadvantage and to their advantage. He considered that while Britain possessed such a pathologist as the President of this Society, and there were many others who would follow his example, there was no need to fear, but there was encouragement to go on and trust more and more to our own resources.

Col. Elliot: Some years back I pointed out that a condition of hypotony may exist for many months, and even for years, in patients who have undergone sclero-corneal trephining, without any evidence that the eyes have suffered thereby. The media remain clear, the vision good, and the visual fields stay in *statu quo antea*. This observation has been confirmed by others, and, not least, by Mr. Collins.

The view that the hypotony following trephining would lead to serious changes was voiced years ago by influential workers, and it was suggested that opacities in the lens would be amongst the evils following the procedure.

More recently cases have been published in which cataract appeared after the operation, and it seems to have been assumed that this complication was the direct result of the hypotony. I would first wish to point out that, because cataract follows, or is taken to follow, the operation for glaucoma, it is not necessarily the result of the hypotony produced or even of the operation itself. We are all aware that cataract may precede glaucoma, or may be a sign of glaucoma, and that under the conditions met with in high-tension eyes, it is not difficult for the lens opacities to be overlooked at the time. Moreover, though the question is too big to be entered into at the present, there are, I feel sure, other conditions beside a direct wound of the lens during operation, which may determine the formation of a cataract. In any case, it is clear, not only from my own experience, but from that of others, that the long existence of hypotony, and I have now been able to keep in touch with cases for six years, does not necessarily cause opacities in the lens. It is open to question whether it ever does.

I submit that the expectation that artificial hypotony would lead to serious changes in the eye, is largely due to a confusion of ideas. All ophthalmic surgeons are familiar with the low tension which follows the suppression of ciliary secretion in serious cases of eye disease. So powerfully has this experience affected some of them, that when hypotony was met with under entirely new conditions they jumped to the conclusion that here again low tension in the eye must be regarded as a serious matter. In ciliary trouble the low tension is a result of diminished ciliary secretion, and is therefore of serious significance, but that is no argument for the belief that low tension, produced by an artificial increase of excretion, should necessarily be taken to be of a like serious nature.

Mr. Collins has referred to the fact that manometer tracings do not show a difference in pressure in the various chambers of the eye, and he clearly recognises that the method is far too coarse to yield results of any value.

The differences in pressure in the chambers are very slight indeed, but it is clear that they exist, or there would not be that flow of fluid through the eye, which we know there is. Such a flow postulates differences in pressure level at different points. The waters of a lake may seem to be at the same level; indeed, the movement in its volume may be so slow that particles suspended in the water appear to be stationary, unless watched for long periods; but so long as we know that there is a stream entering the lake at one end and leaving it at the other, we are confident that there must be a difference, however minute, in the levels of the water at the points where it is fed and drained, and a consequent and corresponding movement of the whole body of fluid. The greatest possible error in connection with the subject has been to consider the condition of the contents of the eye, as though a problem in hydrostatics was involved. Such a hypothesis can lead to nothing but error. The question is one of hydrodynamics and not of hydrostatics.

Mr. Collins has spoken of the changes in the depth of the anterior chamber under conditions of disease, as indicating a difference in pressure between it and the vitreous chamber, and has mentioned the failure of manometer readings to give similar indications in healthy eyes. I wish to lay strong emphasis on a point on which I am sure I shall have Mr. Collins's support, viz. that the manometer is far too coarse an instrument to give reliable indications of the minute differences of pressure which undoubtedly exist in the various chambers of the eye. There is a far subtler physical indication that such a difference in pressure exists, which we learn from a consideration of the very interesting observations made by Ulbrich of the case in which a coloboma of the iris was filled in by a delicate transparent membrane, behind this the fluid in the posterior chamber could be seen to accumulate, until it acquired sufficient pressure to burst its way through the pupil. As it did so the bulging membrane dropped back again, the action being repeated

at irregular intervals and being influenced by the pressure of the lids and by other factors. Many years ago I saw a similar case in Madras, in which the living manometer was furnished by an atrophic patch in the iris, the result of an old inflammation. The bulging of this patch under pressure from behind and its subsidence at intervals could be easily watched. Unfortunately I did not at the time appreciate the value of the opportunity afforded me, and beyond demonstrating it as an interesting phenomenon, I regret to have to confess I threw away the chance of a lifetime. These observations prove a difference in pressure between the anterior and posterior chambers. Now, inasmuch as the fluid from the vitreous must presumably pass through the posterior chamber to reach the anterior, it is obvious that the pressure stands at the highest level in the vitreous chamber and at the lowest in the anterior chamber, the posterior being intermediate between the two. This is self-evident, for otherwise no flow could take place.

I venture to think that no suggestion in Mr. Collins's helpful paper is of more interest than in regard to the transudation of serum into the anterior chamber from the surface of the iris, the posterior chamber remaining free of the pathologic exudate. We know that the iris crypts are credited, under normal conditions, with helping to remove the aqueous fluid from the anterior chamber, but the idea that the current can be reversed is of the greatest value to anyone who is interested in the study of the factors which influence the maintenance of intra-ocular pressure by means of the fluids secreted within the eye. That what has always been regarded as an excreting structure, should prove on occasions to be a secreting surface (using the word secreting in its widest sense), is so surprising that it makes us pause to reconsider our fundamental conceptions of the subject. It has long been known that the secretion poured out, after emptying the aqueous chamber, is abnormal in quality, but so far as I am aware it has always been assumed that the source of this altered fluid is the ciliary body.

The observation that the coagulated fluid is collected in the anterior division of the aqueous chamber alone furnishes one more interesting confirmation of the accepted view that the flow of lymph in the eye takes place in a forward direction from the posterior to the anterior divisions of the chamber; otherwise the exudate would inevitably have been carried into the posterior cavity.

With regard to Mr. Collins's observation of the transitory tendency towards myopia after trephining, the condition is one which I have very often noted. It is met with, as he says, in cases of shallow chamber, and I have seen as much as two and a half diopters of myopia within a fortnight of the procedure. As the flap consolidates the chamber deepens, the lens system recedes, and the myopia disappears, the eye gradually returning to its original state of refraction. I have always explained the phenomenon to myself as being due to the advance of the lens system. Mr. Collins suggests that a second factor may be that the diminution in the tension of an eye may cause relaxation of the suspensory ligament and so allow the convexity of the lens to increase; but I would suggest that the advance of the lens would counteract some of this tendency by automatically increasing the pull of the suspensory ligament.

Mr. Johnson Taylor desired to mention one clinical point with reference to detachment of the choroid. Some years ago he had a case of glaucoma, and he operated upon it, doing, or trying to do, a trephining operation. Some days afterwards, when examining the patient with the ophthalmoscope, he was horrified to discover what appeared to be a growth. It occurred to him that he had made the great mistake of regarding it as a simple chronic glaucoma, whereas it was glaucoma secondary to a growth. After a time, however, it quieted down, the detachment disappeared, and the supposed growth vanished.

There was another point he wished to mention, and on this he would like to ask Col. Elliot a question. Two or three years ago he wrote asking that gentleman what was

the proper thing to do in a case of scleral trephining which had resulted in a constant and severe lowering of tension. He received no reply, and ultimately he referred the case to the President.

Mr. Herbert Parsons considered that the Society was very greatly indebted to the President for having directed attention to hypotony: it was a subject which had been much neglected in the past. Throughout the literature one found constant references to increased intra-ocular tension, but relatively little attention was drawn to the subject of diminished tension. Mr. Collins had done a great work in drawing special attention to the latter, and in such a scientific manner.

He did not agree with Col. Elliot as to the probable slight results of continued diminished tension. He thought it probable that the results in long-continued lowering of tension were as bad as, though more delayed than, those following increased tension, and the President's paper pointed entirely in that direction.

The President said the case mentioned by Mr. Johnson Taylor was one of those he had in mind when he referred to the possibility of minus tension persisting for a year with perfect vision. That, as Mr. Parsons had suggested, was not what he would have expected as the result of his pathological findings.

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2. *Sclero-corneal trephining for hypertony: an experience of some two hundred and fifty operations.*

By J. Gray Clegg.

In medicine, as in other spheres of thought and action, fashion has a certain sway, but, undoubtedly, until further knowledge accrues *re* the real nature of glaucoma, and therefore the best method of preventing or curing hypertension, the newer procedures by which the disease is being treated, securing a filtering cicatrix, has come to stay.

Of the various types of operation suggested to secure this safety valve, that of sclero-corneal trephining is one of the simplest and most satisfactory. In the early days, after the introduction of any new method or remedy, exaggerated claims are made for it, and too favourable a view is often taken of its effects, and, therefore, of its future possibilities.

I consider that, from the cases dealt with by me by this method of trephining, it will be seen that it is not a panacea, but if I am able to prove that the results are superior to those obtained by the older iridectomy alone, something will be gained. It is interesting in this connection to recall the presidential address of the late Dr. David Little to this Society, delivered on October 17th, 1901. I venture to quote the following paragraphs:

"These 67 cases were traced as far as possible. We found that 11 had died, thus reducing our number to 56. Of the 56 cases we traced 37; the remaining 19 were not to be found.

"Now what was the condition of these 37 cases? Ten had become blind, one from atrophy of the globe, another from a recurrence of the glaucoma. The remaining 8 cases were blind from atrophy of the nerve, the tension in the eight cases being normal. Of the other 27 cases, the vision in 4 of them had considerably deteriorated, and they were slowly going downhill from atrophy of the nerve. In the remaining 23 cases the vision in some was better than at the time of the operation, in most of them it was about the same, in a few it was a little worse, but not in any serious degree. Upon the whole they appeared to me in a stationary condition, and perfectly satisfactory.

"Twenty-three cases out of 27, or 62 per cent., have held their ground seven to twelve or thirteen years after operation—a result that I would consider quite favourable among the poor class of hospital patients. I noted, with the one exception above mentioned, that all had a normal tension."

These remarks of Dr. Little related solely to chronic

simple glaucoma, and I consider that they could not be beaten anywhere, for he was an ideal operator as an iridectomist.

The figures that I shall refer to later deal with some 204 patients, on whom the trephine operation was performed 259 times.

Operative Technique.

The method employed differs but little from that described by Col. Elliot. Local anæsthesia was used in every case. It is produced with a 4 per cent. solution of cocaine hydrochloride. After the instillation of two drops at a short interval, one or two ophthalmic tabloids of hemisine are introduced into the conjunctival sac and the lid kept closed for two minutes. After blanching of the eye the patient is directed to look towards his feet, and then, with a spatula, cocaine crystals are spread on the sclera just above the limbus at the site selected for the trephine hole. The upper lid is held up by the finger for the interval of one minute, so that the crystals, only slowly dissolving, are not displaced. By this means I have found it possible to operate in almost all cases without any severe pain being experienced by the patient. After the release of the upper lid the conjunctival sac is again washed out and the speculum introduced. Next a long incision is made by sharp-pointed scissors at a distance of 8 to 10 mm. from the cornea, curved concentrically with the limbus. The flap, consisting of the conjunctiva, which intervenes between the incision and the corneal limbus, is raised by cutting the subconjunctival tissues with blunt-ended scissors, curved on the flat, the concavity being always towards the globe, until the bluish corneo-scleral junction is reached. Next the so-called splitting of the cornea is proceeded with by scratching with the closed points of curved, sharp-ended, ordinary iris scissors. A few strokes bring the points up against a dense overhanging edge, which is formed of the more superficial layers

of the cornea itself at its junction with the conjunctiva. Now the bluish tint of the cornea has become much more evident, and it should be 1 to 1·5 mm. in breadth. In order to ascertain the exact position, I frequently lay the point of an iris repositor against the overhanging edge, flat on the sclero-cornea, and then lay back the conjunctival flap, which has been held by the left hand with fine, horizontally ribbed, conjunctival forceps. The exact extent of the splitting of the cornea is then evident through the transparent flap. If, then, I am convinced that an opening in the dense sclero-cornea will definitely enter the anterior chamber, I again draw down the conjunctival flap, exposing the sclera, and apply, at once, a 2 mm. trephine, pushing the side of it against the overhanging edge. Having applied the trephine and rotated it once, the forceps holding the flap are rejected, and with fixation forceps the conjunctiva at the junction of the flap and the portion remaining normally attached to the limbus is firmly held. Thus a good hold on the globe is secured during the further rotation of the trephine on its long axis. If there is any doubt as to the position of the cutting edge, the trephine can be withdrawn temporarily, and the conjunctival flap put into its normal position, showing at a glance whether the circular incision will cover the anterior chamber margin. For the first rotation or so, in order to get a good "bite," the trephine is held vertically to the globe, but, this having been secured, a definite inclination of the distal end of the instrument towards the optic axis is made, and the rotation continued. I always endeavour to penetrate the thickness in the proximal half only, leaving the upper portion of the disc attached to the sclera in the deeper layers.

The thickness and toughness of the sclera varies considerably, and much depends on the sharpness of the cutting edge of the trephine. It therefore follows that it is not always possible to estimate when the anterior chamber has been entered with absolute certainty. Sometimes a definite sensation is felt by the fingers on pene-

tration, but, of course, one is always sure of the fact if aqueous escapes—sometimes in sufficient quantity to allow conspicuous shallowing of the anterior chamber. Not infrequently, however, the patient gives a slight start or makes an expression of pain; any of these experiences indicate perforation. The trephine is then withdrawn, and, unless the patient is remarkably quiet, a knuckle of iris presents in the opening. This is seized by straight iris forceps and a small portion snipped away.

No traction on the iris while in the grip of the forceps should be made, otherwise the opening may become blocked by iris tissue in spite of excision of a portion. Immediately after the snipping the iris usually retracts, and a small peripheral coloboma appears, which, however, is not so evident as later, when full retraction of the iris has taken place. It is not always possible to estimate, accurately, the size of the piece excised, and therefore the extent of the coloboma produced varies in different cases.

I do not consider it essential to success, to perform an iridectomy, in those cases where the iris does not prolapse, and the aqueous escapes without ballooning up the posterior chamber. I omit it sometimes. However, in my opinion, a small coloboma give a greater sense of security to the operator against subsequent blocking.

Now and again the iris repositor has to be used to get the pillars of the coloboma away from contact with the scleral wound. When the iris has been dealt with I seize the proximal loose portion of the disc by straight iris forceps, and endeavour to cut away one-half to one-third of it only. It is essential, however, to be certain that the posterior lip of the disc, including a portion of Descemet's membrane, is taken away. In one recent case I had a return of tension in an eye within five weeks, although the eye had been subjected to instillations of a strong solution of atropine for the first few weeks after the operation without rise of tension. I could find no fault with the operation, but came to the conclusion that a thin portion of the posterior layer had

been left, which, after the lapse of some weeks, healed and prevented filtration. Anyhow, the repetition of the operation, opening up the same wound, put the eye in a perfectly satisfactory condition.

When the iris is well retracted, the conjunctival flap is replaced, and then three very fine black silk stitches are introduced, closing the original conjunctival incision. The upper part of the conjunctiva retracts very considerably under the upper lid, and, on attempting to seize it, frequently the fine-ribbed forceps pull down subconjunctival tissue or Tenon's capsule, which looks like conjunctiva; but with care the real layer can be secured and recognised by its smooth shiny surface.

Lastly, I wash out the conjunctival sac with sterile saline solution, and instil at once several drops of a solution of atropine sulph., gr. iv ad. ℥i. The bandages are applied, and if the patient remains comfortable the eye is not looked at for two days. I then expect to find the anterior chamber formed, and the pupil fairly well dilated. On the fifth to the seventh day I remove the conjunctival stitches, and if all is well, the patient is ready for discharge on the tenth day.

The thickness of conjunctivæ and subconjunctival tissues differ very considerably. I think it wise to get as much tissue as possible when nearing the position of the trephine hole.

I strongly prefer the large conjunctival flap described, for, undoubtedly, it allows of better filtration, and less likelihood of the formation of a large and irritating bleb at the site of the trephine hole. The size of the bleb is certainly less by the method of operation I now employ.

Buttonholing the conjunctival flap very rarely occurs, even with the thinnest membrane, but care should be taken to avoid tearing it by the forceps which hold it. In some cases the flap has been held in position overlying the cornea by a twisted pledget of wool soaked in the antiseptic solution employed.

The term "splitting of the cornea" is sometimes used,

but in one or two cases where I have been specially anxious to get far forward, I have taken measures to include in the conjunctival flap some of the superficial layers of the cornea, thus truly splitting it, but I find that if the opening is covered by such true corneal tissue firm healing takes place, and no filtration is possible.

I have tried several sizes of trephine, namely, 1, 1·5, 2, and 2·5 mm., but very shortly after having taken up this operation I came to the conclusion that the 2 mm. trephine is far away the best, for the following reasons:

(1) An opening of this size allows ordinary iris forceps to be freely introduced even, if necessary, into the anterior chamber to seize the iris.

(2) As I only remove the anterior portion of the disc, the eventual opening is more likely to communicate directly with the anterior chamber.

(3) The risk of prolapse of the root of the iris or of the ciliary body is greatly diminished.

(4) The superficial extent of the opening when only one-third to one-half of the disc is removed, is not greater than if the 1·5 mm. trephine is used and the whole is taken away, as is frequently practised.

Kind of trephines.—I have tried several forms of trephine instruments, but quite recently have employed that suggested by Harrison Butler, and I now consider it to be certainly the best. It is longer than most others, and allows the fingers rotating the instrument to be so far away from the wound that the latter is easily visible during the rotation; whereas some of the others, when used, have prevented this clear view.

Secondly, the roughening of the distal end gives a perfect grip; and lastly and most important, one excursion of the ball of the thumb and that of the index finger produces several revolutions of the trephine, and, therefore, more rapid opening of the anterior chamber, owing to this end having hardly a greater diameter than the cutting portion.

I am asking Messrs. Weiss to make a slight modifica-

tion by making a small hole in the barrel 3 mm. from the cutting edge. This will allow for passage of aqueous as soon as the anterior chamber is entered, and be a further means of ascertaining when perforation takes place.

In all my cases the trephine operation has taken place in the upper quadrant, and even where more than one opening has had to be made in the sclera, the second one has been placed not very far from the original.

In no case where I have personally operated has the trephined disc passed into the interior of the eye.

I have known, at any rate, of one case where the disc left in the anterior chamber led subsequently to serious iridocyclitis.

In perfectly quiet simple glaucoma it is sufficient even to remove but one quarter of the 2 mm. disc. The only form of the disease requiring the whole of the disc to be taken away is where there is a definite tendency to exudative uveitis. Recently, I did the operation in a patient suffering from old disseminated choroiditis, and had to repeat the surgical procedure four weeks later.

In two cases an extraction of cataract has been done at a considerable period after trephining. The section of the cornea was made in the usual position above, cutting across the trephine hole and obtaining a conjunctival flap. No interference with recovery was noted, but in one of these, however, the tension rose, and the eye became blinded. In the other, after a discission some months later, vision of $\frac{6}{18}$ J. 4 was obtained.

Where the complete formation of the anterior chamber has been delayed beyond the usual time of two or three days, I have found sometimes that the chamber became normal after the removal of the stitches. Once, however, during the removal of the stitches, the anterior chamber gave way, but it reformed by the following day.

Hæmorrhage into the anterior chamber during the operation has been noted several times, especially in the subacute and acute form, but it has given rise to no trouble. No special steps were taken to wash out the anterior

chamber. In a few cases no hæmorrhage was seen at the operation, but blood was found on the first dressing.

The length of time of recovery was always longer in the acute form, but the longest I have ever had to deal with was in an old man with simple glaucoma. Recovery was retarded by persistent secretion from the Meibomian glands without definite suppuration. The globe itself was not affected, and vision was quite as good a year and a half after the operation as before.

Number of Patients, 204.

Total Number of Trephinings on above, 259.

(A few cases have not been included owing to insufficient records.)

Age Incidence.

0– 9 years	3
10–19 „	1
20–29 „	1
30–39 „	15
40–49 „	31
50–59 „	64
60–69 „	66
70–79 „	12
80–89 „	9

Age not recorded, 2.

Both eyes operated on in 52 patients.

Side Affected.

Right	126
Left	125

Types of Cases.

Glaucoma, acute	23
„ subacute	22
„ chronic	186
„ absolute	2

Glaucoma, hæmorrhagic	1
,, traumatic	3
,, secondary	7
Buphthalmos, congenital	1
Retinitis pigmentosa	1
Sympathetic disease	1
Staphyloma, anterior	4

The secondary cases include 1 of aphakia, 3 of cyclitis, 1 of neuro-retinitis, and 1 of embolism of central artery of the retina.

In 6 eyes the operation was performed twice, and in 1 thrice, owing to recurrence of the hypertony.

Three eyes were, on account of recurrence of tension, operated on months or years after an excellent iridectomy.

In one case Lagrange's sclerectomy had been done to the other eye, and in another an iridectomy had been done previously to the other eye.

In one instance, in double subacute glaucoma, I did a trephine on one eye and iridectomy on the other at the same sitting in an elderly patient. Here the iridectomised eye settled down the more readily. In fact, I am now rather of the opinion that iridectomy is advisable for acute and subacute glaucoma occurring in old, debilitated subjects, and it is for these alone that I reserve the older operation.

I have not, as yet, attempted the trephine operation in a case of conical cornea as suggested by Col. Elliot.

Size of Trephine used :

1 mm. in 1 case, 1·5 mm. in 25, 2·5 mm. in 5, and the remainder 2 mm. Since January, 1914, the 2 mm. trephine has been employed in every case.

An accompanying peripheral or complete iridectomy is recorded in 203 of the cases, but in only a very small proportion was the coloboma confluent with the original pupil. In ten cases loss of vitreous occurred, but all these were in the early days. Vitreous is never seen, or never should be seen, unless, indeed, where a trephine is

done for hypertension following dislocation of the crystalline lens. The glaucomatous condition was associated with retinal hæmorrhages in four cases. I regard trephining, however, as being far superior to iridectomy in the hæmorrhagic form, the risk of profuse intra-ocular bleeding being much less. In three cases old standing choroiditis was present. A few days after the operation in quite a small percentage a separation of the choroid has been noted, but always it has returned to position without any special treatment after the lapse of some weeks. According to the hospital notes the separation of the choroid only occurred in two cases, but separation of the retina in eight. My own view is, however, that the greater number of these were really separation of the choroid; but in one there was undoubted detachment of the retina before the operation.

It has always been held that any surgical interference with an eye suffering from sympathetic disease is almost always fatal, but where persistent high tension is found I think trephining is a safe procedure. In one case noted no exacerbation of inflammation took place, and although the vision eventually failed, the tension remained normal ever after; the unfortunate result being in no way attributable to the operative interference.

For retinitis pigmentosa I have tried the operation once only, and that in a very advanced case, with mere perception of light before the operation, but this disappeared, no doubt, from further progress of the degenerative changes, and could not be attributed to the trephine operation.

The one case of congenital buphthalmos in a baby certainly appeared to be quite satisfactory.

For a time after trephining the intra-ocular hæmorrhages met with in the one case of hæmorrhagic glaucoma were not more marked, but later the vision was lost by a large hæmorrhage taking place at the macula.

In anterior staphyloma four eyes were operated on. Three were not improved, and one was doubtfully improved.

There were three cases of iridocyclitis. One was improved, one remained stationary as regards the cyclitis, but of the third there is no record.

After the older operation of iridectomy for glaucoma, very frequently further loss of vision after the operation, took place; it was ascribed to progressive atrophy of the optic nerve. According to my records I only find two, among the whole number traced, in which loss of sight could be attributed to this cause. On the other hand, in two eyes cupping of the disc disappeared after the operation.

As regards the fields, the records are not sufficiently satisfactory to make any very definite statement. In my opinion, for the most part they were much the same after operation as before. In two cases, however, further contraction took place on the temporal side only, and in one case marked contraction of the whole circumference set in. On the other hand, in two patients some slight enlargement of the field was found. All these refer to cases of simple chronic glaucoma.

In five of my cases the eyes were subsequently removed. One was an anterior staphyloma, two where shallow anterior persisted and the tension remained up, and two were lost by acute panophthalmitis, one at an interval of twelve months, and another at the interval of two years.

The fear of subsequent panophthalmitis certainly has deterred many surgeons from resorting to the trephine operation; but the recurrence in less than 1 per cent. of my cases does not appear to me to be a sufficient reason for discarding an operation which, in other respects, is so satisfactory.

The loss of an eye by acute ophthalmitis is, of course, very striking, and makes a deeper impression on the mind of the surgeon than a large number of cases whose sight slowly decays after iridectomy from progressive atrophy of the optic nerve, or from other complications.

It was always the practice in old days to remove eyes painful with absolute glaucoma, but such a cosmetic loss, I think, may be prevented by the trephine operation, as is

proved by two of my patients, who have retained their own—though sightless—organ.

In any case where there is a return of tension at a considerable period after the original trephine operation, I advise the use of eserine and massage by the finger through the medium of the upper lid before resorting to further surgical interference.

In the rush of hospital work it has not been found always possible to make readings with the tonometer, but undoubtedly the instrument is of great service, especially in doubtful cases, where the visual acuity is normal, or practically so, and the field shows hardly any contraction, and there is no definite cupping of the optic discs. I do not think the tonometric readings alone justify one in recommending operation, unless there are definite histories of haloes round lights or definite objective signs.

I consider that operation should be resorted to as soon as possible after a definite diagnosis has been made.

To this rule, even, there are exceptions, as illustrated by two cases:

(1) A very feeble old man with undoubted chronic glaucoma, in whom the expectation of life is not long, and under myotica, the visual acuity and the fields are holding their own.

(2) A middle-aged man, whose left eye operative procedure of the older type elsewhere failed to save. The right eye possesses full normal acuity, with a perfect field and healthy disc. Very occasionally he has had slight mistiness of the sight, with haloes for a few hours, but the nightly instillation of a weak solution of eserin has prevented any such symptoms for at least two years. As he is a sensible fellow, and can resort to surgical assistance at once in case of need, I do not feel justified in recommending operation unless examination reveals any incipient failing on the part of the eye to hold its own.

The number of recorded cases with late results, many of which are eyes that have been trephined up to six years, are 96.

As to final results:

	Per cent.
Acute glaucoma improved . .	14 = 70
„ „ stationary . .	5 = 25
„ „ worse . . .	1 = 5
Subacute glaucoma improved . .	9 = 47
„ „ stationary . .	8 = 42
„ „ worse . .	2 = 11
Simple chronic glaucoma improved .	53 = 32
„ „ „ stationary .	79 = 48
„ „ „ worse .	32 = 20

These figures refer to the visual acuity.

With regard to the chronic form, those that were worse were due (1) to recurrence of the disease, requiring further operative treatment; (2) to the quiet iritis which used to be found before the days when I instilled strong atropine solution at the end of the operation; (3) to opacification of the lens, not the result of trauma to it at the operation.

Again referring to the statistics in Dr. Little's presidential address, I have stated that he said 62 per cent. of his cases of chronic glaucoma retained useful vision for years; but in the statistics presented it will be noted that the vision was improved in 53, it was stationary in 79, and only worse in 32—that is, 32, 48, and 20 per cent. respectively.

The best one can expect in most cases of simple chronic glaucoma is to retain the vision which remains at the time of operation, and if 80 per cent. is the true proportion, it is far superior to the 62 per cent. claimed for iridectomy, and I consider in the future, with the experience already gained, the percentage of retention of vision as good as before the operation will be even greater.

I wish to express my obligation to Dr. Lily Stopford for most valuable assistance in the preparation of this paper.

Lieut.-Col. Elliot said Mr. Gray Clegg referred to iridocyclitis as a result of leaving a disc behind in the eye.

Though he, the speaker, had washed out every lost disc, some of the men who came to his clinic to learn left discs inside, and he found that they did more mischief trying to get them out than if the cases had been left alone, so he had insisted on their leaving them. Yet not one of the cases showed iridocyclitis. Why should an aseptic piece of tissue, which was isotonic with the neighbouring tissues, cause iridocyclitis? He suggested to the author that there were other causes of iridocyclitis which were coincident with the disc being left behind. (2) In doing a trephining, there was a danger in stopping too soon. The President had called attention to an extremely important point, viz. that unless a piece of Descemet's membrane were definitely removed, a filtering scar was not obtained; and if one stopped too soon in trephining, one was not able to turn a flap out and get the Descemet's membrane away. There would be a danger of stopping too soon if one had one's tap in the trephine too low. He therefore thought it better *not* to have the tap-hole low down on the trephine as Mr. Clegg had suggested.

The author also spoke of the visual fields. One did not often get a great improvement in the visual fields even in very successful operations for chronic glaucoma. But where there was a congestive element in the case, and operation was done in the congested stage, the case being fairly acute, his experience was that one might count on improvement in the visual fields. He believed that in the chronic cases the atrophy of nerve proceeded at such a pace that one could not hope to improve the visual fields.

Mr. Harrison Butler said that recently he lost two discs, but they caused no harm whatever.

The President said his experience had been similar to Mr. Harrison Butler's; he had lost three discs, and there was no harm whatever.

Mr. Gray Clegg (in reply) said he had seen one case—not his own—of trephine disc left inside the globe which was followed by troublesome iridocyclitis. In no case had

he left the disc in. He did not attempt to cut out the disc ; he only endeavoured to perforate the sclera in the anterior portion, so as to get half the disc out. It was a very important matter to get away the posterior layer. In one case recently, as stated in his paper, tension returned five weeks later, although the eye had stood strong atropine for four weeks, and he, the speaker, could not find fault with the operation. He showed the case to his colleagues, and they concluded with him that the whole thickness of the disc had not been removed, and that a piece of Descemet's membrane must have been left, sufficient for healing to take place across. Almost always, if a third or half of the disc were taken away, provided the posterior layer were included, a good result would be obtained.

XIII. MISCELLANEOUS.

1. *The evolution of the ophthalmoscope.*

By W. H. McMullen.

Like many other important inventions and discoveries, that of the ophthalmoscope was the result of the completion and co-ordination by an exceptionally gifted individual of the work of many previous investigators.

From the very earliest times the glowing appearance of the eyes of certain animals when seen in a dim light must have attracted notice. The phenomenon was referred to by many writers of antiquity, and formed the subject of much vague and fantastic speculation, devoid of any basis of scientific observation until early in the nineteenth century.

Méry (1), in the year 1704, having accidentally held a cat under water, noticed that he was then able to see the blood-vessels and the colour of the retina. He was probably the first man to see the details of the fundus in the living eye. Five years later (1709) de la Hire (2) gave a correct explanation of Méry's observation, showing that immersion under water abolished refraction at the surface of the cornea, and thus enabled the fundus details to be seen. For more than a century after this the possibility of examining the fundus details in the living eye appears to have been lost sight of, and the phenomenon of the glowing eye engrossed the attention of all students. The glow was supposed to be due to spontaneous development of light within the eye, under the influence of the nervous system, and was thought to become more vivid

when the animal was excited. It was not until the year 1810 that it was proved by Prevost (3) to be due to reflection of light from the eye, as it was never visible in a room from which light was completely excluded. In the same year Gruithuisen (4) traced the effect to the operation of the tapetum, and Rudolfi (5) observed that, in order to see the glow, the observer must look into the eye in a certain direction. Further observations on this subject were published by several authors during the next forty years. Among these the work of Brücke is of most importance. In a paper (6) published in 1845 he clearly described the conditions under which the glowing reflex becomes visible, and correctly explained the varying colour of this reflex. He described his method of examination as follows : "I took in one hand a lighted candle, from which proceeded the only light which illuminated the room; in the other I held an opaque screen of paper or wood, and shutting one eye, placed myself opposite the animal, lying in a corner of the room, so that its head lay in the visual axis of my open eye. The screen and candle were held close to the visual axis in such a way that the screen shielded my eye from the candle."

The blackness of the human pupil was a thing that had hitherto attracted little attention, being usually attributed to complete absorption of the light entering the eye by the pigmented coats. In fact, a full explanation of it does not appear to have been given until Helmholtz gave it in 1851 in his treatise on the ophthalmoscope.

A reflex from the pupil of the human eye had been observed in certain abnormal conditions, but the possibility of seeing such a reflex in the normal human eye was not recognised until the year 1846, when W. Cumming, at that time house-surgeon to the London Hospital, published a very notable and suggestive paper (7) entitled "On a Luminous Appearance of the Human Eye." Cumming showed that it was possible to see light reflected from the fundus in the normal human eye by a method of examina-

tion similar to that employed by Brücke in the examination of animals' eyes. He stated the conditions necessary for seeing this luminosity as follows: (*a*) "That the eye must be at some distance from the source of light, the distance being greater in proportion to the intensity; (*b*) that the rays of light diffused around the patient (and sometimes around the eye itself) should be excluded; (*c*) that the observer should occupy a position as near as possible to the direct line between the source of light and the eye examined." He noted the variation in colour and intensity of the reflex with the movements of the eye, and discussed the source of the reflection, coming to the conclusion that it occurred mainly at the surface of the choroid. It is particularly noteworthy that Cumming regarded the possibility of seeing the fundus reflex not merely as an interesting scientific discovery, but as affording a valuable means of clinical examination, as the following passage from his paper shows: "The establishment of the fact of a similar reflection from the human eye to that from the eyes of animals appears to be chiefly important in its adoption as a mode of examining the posterior part of the eye. The retina and choroid, hitherto concealed in the living eye, and little opportunity being afforded of examining their condition after life, in consequence of their diseases not terminating fatally, considerable uncertainty has hitherto attended the diseases ascribed to these structures; but the existence of this luminosity, its non-existence, or abnormal appearance may enable us to detect changes in these structures hitherto unknown, or satisfactorily to see those which we only suspected."

In 1847, a year after Cumming, Brücke (8) described the appearance of luminosity in the normal human eye examined in this way. It appears certain that Brücke made his observation independently, for a few months after the publication of his paper on the subject, having then heard of Cumming's experiments, he published a short addendum (9) acknowledging the priority of Cum-

ming's observations. Brücke, however, made an even nearer approach to the discovery of the ophthalmoscope, as is shown by the following extract from his paper: "When I had shown this experiment to a few of my friends, Dr. Carl von Erlach said to me that a long time ago he had seen, through his spectacles, the eyes of one of his friends glow. He had also questioned me about it once a year before, but we were interrupted and the fact was not mentioned again. He had, however, often since seen the appearance, and had noted that while he saw it his spectacles shone (spiegele). He was able immediately to see my eye glow when I stood before him with my back turned towards the lamp, so that I saw the reflected image of the lamp in one of his spectacle lenses."

It was in the year 1847 that the first ophthalmoscope was invented by Charles Babbage, one of the most distinguished of British mathematicians. Unfortunately, no account of this instrument was published until the year 1854, when Wharton Jones (10) in a "Report on the Ophthalmoscope," published in the *Medico-Chirurgical Review*, wrote as follows: "It is but justice that I should here state, however, that seven years ago Mr. Babbage showed me the model of an instrument that he had contrived for the purpose of looking into the interior of the eye. It consisted of a bit of plain mirror, with the silvering scraped off at two or three small spots in the middle, fixed within a tube at such an angle that the rays of light, falling on it through an opening in the side of the tube, were reflected to the eye to be observed, and to which one end of the tube was directed. The observer looked through the clear spot of the mirror from the other end."

Wharton Jones was a man of high character and great scientific ability, and there is no reason to doubt the accurracy of the above statement, more particularly as he was probably responsible for the failure of Babbage to pursue his investigations. As there is a reference to this subject in Mr. Brudenell Carter's Text-book of Ophthalmic Surgery, I wrote to him a few days ago to ask if he

could tell me anything more about it. Mr. Brudenell Carter, now in his 89th year, very kindly replied to my letter and wrote on April 25th, 1917: "I cannot now remember any further detail, except that Wharton Jones committed the monumental folly of condemning the instrument, so that Babbage threw it aside as a toy of which he thought no more."

While to Babbage belongs the distinction of having first invented the ophthalmoscope, and while we may justifiably take pride in the fact that this achievement stands to the credit of one of our countrymen, that does nor detract from the honour due to Helmholtz as the man who devised and made known to the world the instrument which revolutionised ophthalmology.

Helmholtz's monograph (11) published in 1851, was a masterly work. In it he clearly expounded the optical problems to be solved, and showed the necessity of a method of looking into the eye exactly in the direction of the incident light. He explained how this could be achieved by the use of a transparent mirror, and described a really practical instrument for the examination of the fundus details. The construction of Helmholtz's instrument is probably familiar to all members of this Society. It consisted essentially of a transparent reflector, composed of three thin plane parallel plates of glass, set at an angle to reflect the greatest possible amount of light into the eye, while on the observer's side of the mirror was a holder for a concave lens to enable the fundus details of the patient's eye to be seen clearly. In addition this treatise contained exhaustive studies of various points in relation to the instrument, *e. g.* the advantages of multiple reflecting plates over single, the most advantageous angle of reflection with different numbers of plates, the polarisation of light reflected by multiple plates and consequent diminution of the corneal reflex, the optical principles underlying the formation of the erect image, and the calculation of its magnification. He referred to the possibility of using two convex lenses, in place of a

concave lens, for observation of the image, but did not describe the indirect method as we know it. He noted the insensibility of the optic disc. He indicated the possibility of determining the refraction of the eye with his instrument, and, as showing his appreciation of its clinical importance, I quote the following sentence: "In brief, I believe I may hold the expectation, not to be exaggerated, that all the alterations of the vitreous body and of the retina, which until now have been found in cadavers, will also be recognisable in the living eye, a possibility which appears to give promise of the greatest advances in the hitherto undeveloped pathology of these structures."

There is reason to believe, however, that Helmholtz did not at first fully realise the epoch-making character of his invention. It was von Graefe who first grasped this. Douders (12) in 1886, in the course of an address, on the occasion of the presentation to Helmholtz of the Graefe medal of the Heidelberg Ophthalmological Society, referred to this matter as follows: "He (*i. e.* Helmholtz) never thought, or at least he never said, that the new instrument implied the dawning of a new era for ophthalmology. Von Graefe felt it immediately. When he, for the first time, saw the background of the eye, with its nerve entrance and its blood-vessels, his cheeks reddened, and he called out excitedly, 'Helmholtz has unfolded to us a new world!'"

Helmholtz (13), in his speech on this occasion, gave the following interesting account of its discovery: "The ophthalmoscope has unfolded itself to me simply out of the necessity of discussing, in my lectures on physiology, the theory of emission of light from the eye. Why does the human eye not glisten under ordinary circumstances, since in its background there is situated a spot—small, indeed, but clear white; that is to say, the end of the optic nerve, which must reflect light in the same way as the most sparkling tapetum of animals' eyes? Why do animals' eyes shine with such remarkable lustre, though

they may be only illuminated by a small distant flame? These questions, when once proposed, were not difficult to answer, and now the answer is known to everybody. Once answered, they furnished the means of lighting up the eye of another human being and of seeing it plainly." It appears, therefore, that the discovery of the ophthalmoscope is another example of a result of enormous practical importance following research in pure science, in some degree comparable to the revolution in medicine and surgery which began with the study by Pasteur of the crystals of tartaric acid.

Among the chief defects of Helmholtz's ophthalmoscope were inadequacy of illumination, an inconvenient and time-consuming arrangement for changing the lenses, and the fact that, owing to the distance between the eyes of patient and observer, the field of view of the fundus was small. Nearly all the extremely numerous modifications of the instrument since introduced were designed to overcome to a greater or less degree these various defects.

Improvement in the illumination was secured by the use of a mirror, either with a portion of the coating removed from a small area, or with a perforation in the centre. In 1851, only a few months after the publication of Helmholtz's treatise, Epkens (14), a Dutch mechanic, constructed an ophthalmoscope with a plane mirror, with the coating removed from an oval area in the centre. Ruete (15), in 1852, used a perforated concave mirror, and described the indirect method of examination.

An improvement in the method of changing the lenses was very soon devised by Rekoss, an instrument maker of Königsberg, who modified the original Helmholtz instrument by the addition of two superposed rotating metal discs, each containing four concave lenses of different strengths and a clear aperture.

Innumerable patterns of ophthalmoscopes have since been designed. Every conceivable form of reflector has been employed—plane, convex, and concave mirrors, with

the most various curvatures and diameters, combinations of plane and convex mirrors with convex lenses, silvered lenses, totally reflecting prisms, and the cemented surface of a Nicoll's prism.

As regards the arrangements for changing the lenses behind the sight-hole, one may group the various devices into four classes: (1) Holders or clips into which lenses were inserted singly, as in Liebreich's small ophthalmoscope, which is the most familiar example of this class. (2) Modifications of the Rekoss disc—single, double, or triple discs, or one or two discs with a segment of another. (3) One or more narrow plates with rows of lenses arranged to slide up and down behind the sight-hole. (4) The magazine arrangement devised by Couper, which is also employed, in combination with a disc, in the familiar Morton's ophthalmoscope.

The advantage of being able to approach very close to the patient's eye for direct examination, and at the same time looking perpendicularly through the correcting lens, led to the introduction of various forms of tilting or obliquely set mirrors.

Time will only permit of the bare mention of the considerable class of large fixed instruments usually designated demonstrating ophthalmoscopes. Two examples are exhibited in the next room—Liebreich's (1855) and Priestley Smith's (1874). Instruments have also been designed to enable two or three observers to examine the fundus simultaneously.

For a time binocular ophthalmoscopes enjoyed considerable popularity. The first of these was designed by Giraud-Teulon (1861). It was found in practice that there was little advantage in getting a binocular view of the fundus, and no binocular instrument could be made as simple and convenient to use as a molecular one.

The first electric ophthalmoscope was invented by Dennett, of New York, in 1884. In 1886, Dr. Thomas Reid, of Glasgow, designed an interesting electric ophthalmoscope with a prismatic reflector and a micrometer

scale, an image of which was projected on the fundus. This instrument was shown at a meeting of this Society, but never placed on the market. We have the original model on exhibition here to-day. Another early type of British design is Juler's (1886), which we are also able to show. Most of the recent developments of this type are, of course, familiar to all ophthalmologists.

This brief review of the evolution of the ophthalmoscope is necessarily incomplete. Time will not permit of any description of many interesting types, such as various forms of autophthalmoscope, nor of the recent developments in reflexless ophthalmoscopy, in which Gullstrand's work is pre-eminent; but I trust the short account I have given will suffice to lend interest to the exhibition of ophthalmoscopes in the next room.

In conclusion, I wish to tender my thanks to all those gentlemen who have kindly lent instruments for exhibition at this meeting.

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2. *The apocritic principle and the evolution of visual perceptions.*

By J. HERBERT PARSONS.

WHEN crystals separate out from a solution certain molecules detach themselves from all others of a different kind, group themselves in a particular manner, and produce or create a mass of material which, while retaining essential characteristics of the original molecules, becomes endowed with entirely new physical properties.

When a cat perceives a mouse he receives a vast number of sensory impressions. Visual impressions convey to him the shape and colour of the mouse, and in addition those successive variations in shape and colour and relationship to surrounding objects which constitute the visual impressions of movement. Olfactory, auditory, tactile, and other sensations derived from the mouse have their specific characteristics in relationship to the cat. None of these impressions taken alone or in groups, or even altogether, suffice to constitute the perception of the mouse by the cat. The mouse is perceived as a mouse, and indeed as a particular and unique mouse, only when many or all of the sensory impressions are sifted out of their context and selected and organised into a new complex unit. That unit is a new creation. While it retains many of the essential characteristics of its con-

stituent sensory "molecules" it endows them with entirely new properties. The sensations have been synthesised into a perception, the outstanding feature of which, as emphasised by Prof. Stout, is the "meaning" which it carries with it, and which is truly and scientifically described as being created by the synthesis. The perception is as different from the sensory "molecules" as the crystal from the solution.

When a man talks of mice he thinks of the mouse in general and not of any particular mouse in a particular felt situation. This generalisation or universalisation is a concept. It differs as much from a percept as the latter from a sensation. The concept is born as the result of a similar creative process wherein perceptual "molecules" are segregated and resynthesised.

This creative process, which I call the apocritic principle, is a fundamental process pervading the whole of evolution. It can be analysed, as we have seen, into two consecutive stages—(*a*) a selective segregation and (*b*) a creative synthesis. Doubtless these stages are only an imaginary analysis: they are in reality a single process. So far as we have gone the apocritic principle is little more than a somewhat grandiose term for a schematic description of patent facts. If it has no more value than that our already overburdened scientific terminology might well be spared this further addition. I think, however, that if it is kept in mind as an analytical principle it will be found to possess the truly greater value of a working hypothesis, throwing light on some very obscure problems and perhaps pointing the way to further research. I propose to illustrate my meaning by applying this touchstone to visual sensations and perceptions.

The fundamental property of living matter, from the lowest organism to the highest, may be termed either irritability or movement, according as it be viewed from the subjective or the objective standpoint. Irritability is the property of the organism to respond to stimulus; movement is the objective response. The stimulus with

which we are now specially concerned is light. Light, from the objective point of view, consists of the luminous spectrum and the infinite combinations of its parts. It is, however, to be noted that from this physical or objective point of view, the luminous spectrum is only part of a wider gamut which includes heat on the one side and chemical action on the other, and that both heat and chemical action overlap the luminous field. It is equally important from the subjective point of view to remember that the evolution of the sensory organs is a continual differentiation to subserve stimuli of a more precise and specific nature. Thus, the radiant energy which falls upon the lowliest, merely sentient organism, must arouse in it a simple sensory response which we may conceive as being more allied to our tactile sensation than any other. In other words, the chaotic congeries of stimuli arouses a sentiency which is simple only from want of differentiation, and this in turn gives rise to a simple motor response. As we ascend the animal scale the differentiation of nerve structure is equivalent to the development of specific resonators. Yet, if we bear the apocritic principle in mind, we shall anticipate (1) that the differentiated sensory response will still retain a substratum of its chaotic origin, and (2) that differentiation will be indissolubly bound up with brand new and unexpected properties. By (1) I mean this—that if our surmise is accurate, that the chaotic stimulus produces a sensory reaction of the tactile type, then the tactile element will not be lost, but only submerged in all subsequent differentiations. Doubtless the more precise the differentiation the more submerged the tactile element, yet I think that neurologists will bear me out in the statement that even in the highest visual and auditory perceptions the tactile element is not wholly absent. With regard to (2) I may make the same vast stride to the highest sensory differentiations and point out that the visual and auditory differentiations give rise to the wholly new phenomena of projicience, marking an advance in the potentialities of the animal comparable

only to the later creation of the concept. I need scarcely express my indebtedness to Prof. Sherrington's writings in this connection.

By way of analogy I may mention the differentiation of the cell in its physiological relations. No matter how differentiated a cell may become, it still retains its fundamental cytological characteristics, most obvious in those associated with nutrition, growth, and reproduction. In fact, Dohrn's principle of functional change, the retention of all the fundamental properties of protoplasm by even the most highly differentiated cell, holds good also for those higher functional manifestations with which we are here primarily concerned.

There is reason to believe that the earliest outward and manifest sign of the differentiation to luminous energy is the development of pigment cells on the surface of the body. They are at first widely distributed, and we can scarcely conceive the sensory response as being other than a very slight modification of the tactile sense. If, as is probable, it thus early contains the germ of projicience it must be in a very rudimentary manner, manifesting itself only in the more precise motor response—whether positively or negatively phototropic.

With the development of axial symmetry and metamerism the visual cells, as they may now be called, become aggregated anteriorly and their projicient function becomes accentuated.

When we reach the lower mammals, such as the rabbit, the function of the visual organ is pre-eminently projicient. It is characterised by enormously wide lateral fields. On teleological grounds, if on no other—and they are abundant—it must be conceded that the appreciation of *movement* is the prime function of these organs. The tactile element has become specialised to respond to luminous stimuli. Just as the finger passing over the skin arouses the appreciation of movement, so the light passing over the retina; but whereas one is referred to the immediate environment, the other is projected into

the outer world. The physiological characteristic of each is the successive stimulation of serial end-organs. The psychical equivalent of the tactile stimuli is, first, "something touching," next "something touching which moves." In its primitive form we must regard any other perceived qualities as vague and chaotic, such as we ourselves can only dimly conjecture. With increased differentiation appreciation of size, shape, and temperature are successively born out of the void. So, too, the primitive light sense arouses the vague appreciation of "something bright" and of "something bright which moves." The pigment cells absorb the heat and actinic radiations as well as the luminous, and the transformed energy *must* produce effects—partly nutritive and metabolic—perhaps also effects in consciousness, but if so, effects which are vague and chaotic and undifferentiated. Very early, however, the something bright is a something in the outer world, and moreover in a definite position in space with regard to the organism. Similarly its movement in space is more or less accurately referred to its spatial relations with the individual. Only with increasing differentiation does the something become more or less definitely endowed successively with size and shape and colour, contemporaneously, with ever increasing precision, with more accurate localisation and tri-dimensional qualities.

Let us now turn to the fully-developed visual functions of man, so far as we have yet been able to elucidate and analyse them. It is customary in our text-books to divide visual perceptions into three categories, dependent upon (1) the light sense, (2) the form sense, and (3) the colour sense. This is a convenient and for physiological and clinical purposes a satisfactory classification, except that it leaves out of account the important fourth sense —the sense of movement, which we have seen to be primitive. Obviously, so far as visual psychology is concerned, the light sense is the fundamental and most primitive element in consciousness. Applying the apocritic touchstone it is a segregation of those tactile

qualities of delimited areal sensations which are due to a certain group of radiant stimuli from those which are due to other groups of stimuli produced by radiant energy, all of which enter into the production of the primitive chaotic tactile sensation. Owing to the differentation of the nervous structures this selective segregation is not only rendered possible, but their re-synthesis into a new complex is potentialised. This re-synthesis emerges in consciousness as a newly created light sense which, while retaining the areal characteristics of the primitive tactile chaos, has become endowed with the functions of projicience and the discrimination of light and shade.

The same touchstone may be equally applied to the form sense. The primitive chaotic tactile sense already possesses dim adumbrations of form or areal delimitation—this is the really primitive form sense, and it has no visual properties in consciousness. The second stage in its visual development is the vague sense of form which accompanies the visual perception of a bright, moving object on a dark background or *vice-versâ*. But the term "form sense," as commonly understood and applied, refers only to the precise discrimination of form by central vision. This is the third stage of the development of the form sense, and itself is divisible into degrees of lower and higher development. In this third stage structural differentiation and concomitant functional development—the type and arrangement of the cones in the macula on the one hand, and the increased precision of temporal and spatial induction, etc., on the other—so alter the segregated elements and their final re-synthesis that a hitherto unknown precision in the perception of areal delimitation is acquired. At a later stage this newly acquired "sense" is correlated with other newly acquired psychical attainments; these interact with and facilitate each other, so that the perception of letters, etc., is facilitated. It is these higher grades of the still so-called form sense which are investigated in the clinical estimation of visual acuity.

Throughout its evolution and development the form

sense retains all those characteristics of the primitive tactile sense which have been segregated by the light sense, and, moreover, it also retains them in the re-synthesised form in which they appear in the light sense.

The evolution of the colour sense precedes in order of time the third stage of development of the form sense. Many theories, none of them wholly satisfactory, have been advanced to account for the phenomena of colour vision. Complex as these phenomena are, they undoubtedly show that the number of "resonators" which have been differentiated to subserve them are far fewer than the innumerable diverse stimuli which produce them. Whatever may be the difference of opinion as to the segregation of the stimuli and their re-synthesis there can be no doubt as to their occurrence; and here, as in the form sense, all the features of the light sense are present, with others incorporated, and not merely added.

My chief object in drawing attention to this method of viewing visual perceptions is to show that it helps to explain certain difficulties.

(1) *Central vision.*—It has been shown by Riccò, and confirmed by Charpentier and others[1], that in order to produce a luminous sensation at the fovea the total quantity of light, *i. e.* the product of the area and the illumination, must attain a certain value, and that that value is constant for a given condition of adaptation. Here we have a precise mathematical law, and it is associated with the most highly differentiated structures. Asher found that for the range of light intensities used by him, the apparent size, up to a visual angle of 2′ to 3′, depends entirely on the quantity of the light. By varying the area and the intensity of the light smaller areas may be made to appear larger than really larger areas. It is well known that the apparent size of points of light varies with the light intensity. The so-called "magnitudes" of the stars depend upon this fact.

Similarly with regard to colours, Charpentier showed

that the chromatic threshold also depends upon the area stimulated ; and it can be readily demonstrated that the hue changes as the area is altered. As we should expect, for the more highly differentiated colour sense, the law associating area stimulated with intensity is not so simple as for white light. Further, as Abney has pointed out, as the intensity of the light is diminished gradually to extinction of the sensation, a coloured square or disc becomes first an ill-defined, fuzzy patch of grey and appears finally to depart almost as a point.

It appears to me that these liminal stimuli reproduce the characteristics of primitive chaotic sensations, in which size and colour and other qualities are as yet undifferentiated.

More instructive are the threshold values of so-called visual acuity(2). If we take as our criterion the discrimination of two points of light as disparate, the minimum visual angle is little less than one minute. A very much smaller difference of position can, however, be easily recognised. Hering showed that if a black and white surface are separated by a vertical line, the lower half of the black surface being movable over the white, a movement of 10″ can be discriminated. Hering therefore distinguishes between the visual sense of position (optischer Raumsinn), the visual resolving power (optischer Auflösungsvermögen), and the form sense (optischer Formensinn). Three corresponding criteria have been suggested: (1) Minimum visibile, (2) minimum separabile, and (3) minimum legibile or cognoscibile. The enormous effect of variations in the illumination on the discrimination of test types is well known. Throughout it will be found that the form sense, whatever its stage of development and whatever the criterion, becomes merged at the threshold into a primitive type of light sense, in which the qualities of form, colour, etc., cannot be differentiated.

The obliteration of the discrimination of form and colour at extremely high intensities of illumination is perhaps to be explained in the same manner.

(2) *The field of vision.*—Enough has already been said to show that the very common practice of calling the field for white the field for form is much to be deprecated. It is true only of the form sense in what I have called above the second stage. But the form sense in ordinary parlance means the capacity for distinguishing complex forms, such as letters, etc., and this is practically absent even under normal conditions with ordinary perimetric observations. Dor has shown that visual acuity is reduced to $\frac{1}{15}$, 10° around the point of fixation, and is practically negligible beyond.

With regard to colours it is true that with intense degrees of illumination they can be distinguished almost, if not quite, to the extreme periphery. Under normal conditions, however, there is a peripheral totally colour-blind zone, separated from the central trichromatic area by a dichromatic zone. Now, it is well recognised that the periphery of the retina is particularly sensitive to the appreciation of *moving* objects. For teleological reasons it has retained this primitive characteristic in a very pronounced manner. A careful consideration of the functions of the peripheral part of the retina (including, of course, the whole of its nervous mechanism—central as well as peripheral) shows that we have here in its least differentiated form the primitive chaotic light sense, with its predominant feature—capacity to appreciate movement—least submerged. From this point of view we should expect that, in recovery from lesions which abolish peripheral parts of the field, the appreciation of *movement* in objects would be first restored.

There is evidence that such is the case. Wilfred Harris([3]), in 1897, pointed out that in cases of transient hemianopia following epileptic attacks he had observed that the appreciation of movement of objects returned before that of "form" or colour. Recently, G. Riddoch([4]) has brought forward still more conclusive evidence, derived from war injuries of the occipital lobes. From the preliminary account of his observations he concludes

(1) that the consciousness of "a something moving" should be recognised as one of the visual perceptions; (2) that it may be dissociated from the perception of a stationary object; (3) that where recovery of vision occurs, the perception of "movement" precedes that of the object; and (4) that recovery of movement vision begins at the periphery. He expresses an opinion that a wider conception of what vision is might be obtained if it were considered, not as a special sense, but as a part of general sensation. His cases indicate some visual defects which resemble disturbances of sensation, such as: (1) Dissociation of primary visual perceptions of light, movement, stationary objects, form, and colour; (2) inability to localise an object seen and to estimate its length; (3) inability to appreciate "difference"—relative lengths and distances; (4) inability to distinguish between a flat disc and a sphere.

It appears to me that Riddoch regards these dissociations too narrowly from the purely pathological point of view. The wider conception of what vision is, which he very rightly emphasises, can only be attained if these dissociations are regarded from the broad standpoint of evolution. It will then be found, as I have tried to indicate, that they are always *relative*, whether viewed as it were in longitudinal or transverse section. In longitudinal section we have the apocritic process at work differentiating each perception in turn from the primæval inchoate—broadly speaking, "tactile"—sensation or perception. Here the differentiation is eminently adaptive and practically effective, yet never complete, but only relatively dissociative. The same general characteristics appear in transverse section—the physiological condition of the individual animal—no matter what the stage of evolution. In the highest stage, man, the same relative dissociations appear, fusing into the primitive chaos at the low intensities of scotopic vision and in the extreme periphery of the field, selectively segregating in various degrees at the higher intensities of photopic vision, all fusing again

into quasi-pathological phenomena at the highest intensities.

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3. *The value of the ophthalmometer in estimating the refraction of the eye.*

By Ernest Clarke.

Table I.

2500 Individuals whose sight after correction was normal and who had no disease of the eyes.	(1) Same refraction in both eyes (657)	(*a*) Emmetropia (see Presbyopia below)	9
		(*b*) Hypermetropia	63
		(*c*) Myopia	22
		(*d*) Astigmatism—	
		Hypermetropic	438
		Myopic	113
		Mixed	12
	(2) Refraction different in the two eyes (Anisometropia)		1843
			2500
5000 eyes (as above)—			
Emmetropia			56
Hypermetropia			425
Myopia			216
Astigmatism			4303
			5000

Of the 2500 individuals, 961 were presbyopic, and only 9 of these were emmetropic.

Table II.

500 consecutive refractions = 1000 eyes.

Astigmatism	Under 1 D.	Under ·5	·12	164	542
			·25	306	
			·37	72	
		·5 and over	·5	108	192
			·62	14	
			·75	70	
	1 D. and over				132
No astigmatism					134
					1000

Ophthalmometer correct = 982.

A recent long illness gave me the opportunity of making a statistical examination of a large number of refraction cases; I examined 10,000 eyes. I selected only those cases where both eyes were used, where vision was not worse than $\frac{6}{12}$, and where the refractive error was the only trouble. I have embodied the results of my examination of the later 5000 in Table I, and a more recent selection of 1000 consecutive refractions in Table II.

It is not my purpose to describe ophthalmometry or the various instruments in use; suffice it to say, that the ophthalmometer or astigmometer is an instrument for measuring the astigmatism of the cornea, and that I cannot speak too highly of the latest model made by Meyrowitz, of New York, which, in my opinion, and from almost thirty years' experience, stands pre-eminently the best on account of its simplicity and accuracy, and the ease and expedition with which its readings can be recorded. I have heard some say that they have tried the ophthalmometer, and discarded it because it was of no help, and that the readings disagreed with the examination by retinoscopy and before the type. They may have been using an unreliable instrument, or, what is more likely, they probably had not the patience to master the details.

There are two very important points to remember in using the ophthalmometer: (1) The patient's eye must be

focussed most accurately in the centre of the tube, the smallest deviation from this giving a wrong reading; and (2) the lids must be in their natural position, *i. e.* the patient must not be told to open the eyes widely or lift the upper lid; and lastly, each instrument has some special characteristic of its own. In the instrument I now use I have to add ·12 inverse astigmatism to the reading, *i. e.* if there is recorded ·12 direct astigmatism, astigmatism is absent, and if there is ·12 inverse astigmatism recorded, ·25 is present.

A very short use of this instrument enables one to master these details.

Taking the ophthalmometer readings of the eyes in Table I, I find that they were useless in 139 cases out of 5000, *i. e.* 2·78 per cent., but that in 97 per cent. they were most valuable, and in some cases so valuable that no other method of examination could have replaced the ophthalmometer.

In Table II the more recent cases, where I have been working with the most recent model and have naturally gained experience in manipulation, I found that in 1000 cases only 18 readings were useless, that is, 1·8 per cent.

The longer I practise the more certain I am that the astigmatism of the cornea is, in the large majority of cases, the only astigmatism, the presence of which we want to ascertain. Any lenticular astigmatism that is present is, in most cases, dynamic and corrective. In a very few cases the lenticular astigmatism is static, and then, of course, the ophthalmometer is useless; but we see, from the figures above, that these cases are certainly not more than 2 per cent. of those examined, as a few of the cases where the ophthalmometer is useless show the presence of *irregular* astigmatism, which naturally vitiates the record.

Therefore, the ophthalmometer is valuable in direct proportion to the prevalence of astigmatism.

In Table I, no less than 4303 out of 5000, and in

Table II 866 out of 1000, *i. e.* is 86 per cent. of the refractions, astigmatism was present. The preponderance of these are low errors. We see, in Table II, that out of the 866 no less than 542 were astigmatism of less than ·5 D.

Now, the correct estimation of these low errors, both as regards amount and axis, is of vital importance if we want to cure the countless functional disorders they produce, and it is exactly here where the ophthalmometer comes in. I defy the most skilful examiner to diagnose the presence of, say, ·25 of astigmatism 5–10° from the horizontal or vertical, with certainty. Even with the patient under the influence of a cycloplegic it is difficult sometimes to obtain a definite answer, but once armed with the knowledge the ophthalmometer gives, we can prescribe the cylinder with a certainty that if the glass is worn always, complete relief from the symptoms will soon be obtained.

Apart from the saving of time in the examination, the confidence that the ophthalmometer gives is priceless, and I have, I think, proved by my figures that this confidence is warranted in 98 per cent. of the refractions that one has to treat.

I can add no more, except to implore those who desire to do their refraction work well and so relieve innumerable troubles, to give the ophthalmometer a fair trial, and I am confident they will soon be as loud in their praise of this valuable instrument as I am.

Mr. Arnold Lawson said he had used ophthalmometry, but he thought it deplorable that such a lot should be made out of minute astigmatic errors. Doubtless they had their value, but to regard a ·25 error as one for glasses to be worn constantly seemed to him to be deplorable. That was treating the matter as if there were some sort of disease present, or as if anything would happen if glasses were not constantly worn. The presence of a small degree of astigmatism was, in his opinion, of no value

whatever unless it caused symptoms. When it did produce symptoms, it had the value which was measured by those symptoms. If a man with a small degree of astigmatism had only occasional headache, why should he be forced to wear glasses all day, when correction for reading purposes alone would suffice to relieve symptoms?

Sir GEORGE BERRY said he should like to confirm everything that Mr. Arnold Lawson had said. In his opinion far too much could be made of small optical defects.

Some members would remember that when the ophthalmometer first came into use there was an idea, now known to be erroneous, that certain symptoms met with in astigmatism were ascribable to the effort by which accommodative changes in the lens could counteract a certain degree of corneal astigmatism. He referred to this now, because Mr. Clarke used the term "corrected" in connection with the crystalline lens. There was no such thing as correction of any corneal astigmatism by active lenticular changes.

Mr. ROWAN said that in using an astigmometer, and working out retinoscopy afterwards, if one found astigmatism with the astigmometer and the patient did not improve, he did not recommend forcing cylinders on the patient. Astigmatism was, in many cases, corrected when there was no need. If the patient refused cylinders, one would often find, by working out retinoscopy, that there was no real astigmatism present. He used the astigmometer, but did not rely upon it entirely. In the *British Medical Journal** he published a paper in which he gave results in private cases of astigmatism, both corneal and absolute, worked out by retinoscopy, and there was a wide difference between the two. He did not now remember the actual figures.

Mr. CRESSWELL (Cardiff) said that during the last fifteen years he had invariably used the ophthalmometer in all

* *Brit. Med. Journ.*, January 13th, 1912. Out of 1000 eyes examined, the absolute astigmatism and the corneal astigmatism are the same only in 47·5 per cent.

his refraction cases, and he agreed with everything which Mr. Ernest Clarke had said. He always used the simplest form of instrument—one he brought over from Utrecht. He regarded it as particularly useful in children's cases, in which one wanted to determine the exact axis of astigmatism; also in cases in which one did not wish to dilate the pupil, or the media were not clear, and when retinoscopy was difficult, one could, by this means, determine the astigmatism.

Mr. Ernest Clarke (in reply) remarked that Mr. Arnold Lawson said it was deplorable to correct low errors of astigmatism if no symptoms were present. All the cases he referred to, however, were sent to him because of symptoms; they were not people picked out of the street and told to come into his room to be tested! Glasses were given, and the symptoms disappeared.

Sir George Berry and he would never agree on the subject of refraction. He (Mr. Clarke) did not pretend to say what the physiological explanation was, but that there was a dynamic astigmatism of the cornea or the lens—probably the latter—correcting the astigmatism of the cornea he could prove from hundreds of cases. How did one account for the following common example? A girl, say, of sixteen has headaches, and reads $\frac{6}{5}$. She is put under atropine, and when she is under its influence she is found to have ·25 astigmatism in both eyes, with a little hypermetropia. When the cycloplegic has passed off she does not like the correction of the astigmatism because the corrective astigmatism has returned. She is told to wear the correction always and report in a month, which she does, with all the symptoms gone, and the declaration that she sees better now with the glasses than without them.

What was "deplorable" was the prescribing of weak plus spheres, equal in both eyes, for headaches in children for use at near work. These patients invariably have low astigmatism or low anisometropia, or both, which is not diagnosed or treated, with the consequence that the symptoms of eye-strain do not disappear.

4. *Conditions affecting standards of vision in an army.*

By W. Wallace.

In a discussion of the military side of ophthalmology it is necessary to keep in mind certain broad distinctions.

The aim in civil as well as in military practice is efficiency, but the stamp of patient, and more particularly his mental attitude, must not be disregarded. In civil practice the patient seeks advice because he believes he has a visual defect, and will give every assistance to have it remedied. In military practice this is the exception.

The first serious visual test to which a soldier is subjected after enlistment is musketry. He may have passed the test for recruits successfully, but on the range he may complain that he cannot see the sights, or the target, or all three. If a visual defect is disclosed, he may be inclined to make the most of it, and hamper at every turn the efforts of the medical officer who is anxious to render him efficient. Again, it is well known that among certain classes there is a strong prejudice against the wearing of spectacles, and countless hours in the Army have been spent in working out refractions with the point-blank refusal at the end to accept glasses. We have, then, one definite mental factor against us with which it may be impossible to reason, and in the absence of co-operation on the part of the soldier, which in this case is indispensable, the results are unsatisfactory.

Another important point not to be overlooked is the vast difference between peace and war conditions. With a voluntary army in pre-war times the aim was to secure men who were physically fit or capable of being made so after a course of training. Some latitude, therefore, was allowed for the possibility of an immature youth being brought up to a better stage of development after a few months in the Army. This, however, could not apply to vision, and numbers of men, who in other respects were

excellent material, were rejected because they could not pass the sight-test. We have to remember, moreover, that in peace-time the voluntary system prevailed, and men came forward because they wished to become soldiers. It was a hardship to them if they were not accepted, and they were not disposed to obstruct the medical officer in the examination. Thus, men with visual defects occasionally succeeded in slipping through by just coming up to the minimum standard which was then required. The willing recruit was aware that it was in his own interest to make light of or conceal a physical defect, though the examination was of so searching a character that concealment was impossible. It was equally in the interest of the country that there should be no room in the Army for men who were actually, or were likely to become, ineffectives, therefore we could choose the men best fitted for military purposes. With the adoption of a measure of conscription the character of the recruit, in a very large number of instances, underwent a change. He had little of that incentive that made those of the voluntary Army so conspicuous and distinguished in mental and physical qualities. Into the social and political aspects of this question we need not enter, but the contrast between the voluntary soldier and the compelled man will be recalled in scores of instances by every medical officer who has had to deal with troops. I will return to the influence of compulsion upon vision in a moment.

The nature of modern warfare, again, has an important bearing on vision. We are not fighting now as we were in the South African War. In that campaign the soldier had to be able to spot a man a mile off, and, what is more, to hit him. A high standard of vision, therefore, was as essential as powers of endurance. He was always on the move; always incurring the risk of being sniped by some solitary invisible marksman.

We are in the midst of a very different state of affairs now, and the War Office, last year, issued a Letter calling attention to the fact that fighting at close quarters did

not require the vision necessary for long-distance shooting, and that every company officer had his own picked men who were expert with the rifle. It also pointed out that a man who was dependent upon spectacles was at a disadvantage in trench warfare with everything about him wet and muddy, and with little chance of keeping his glasses clean. Thus, not only has the character of our army changed, but also the mode of warfare.

We are now confronted with a host of conditions which in times of peace would be disregarded or deliberately excluded in the aim to produce what may be called a standardised type of man-at-arms. Instead of one type we now have several scores ; instead of a limited number of non-combatant occupations we have multitudes. The enlisted man in the old Army had to be a fighter, whatever else he might be in barracks ; to-day he may still be carrying on his civil vocation or trade, but with the difference that he is under military discipline and in uniform. Obviously, this includes those who hold temporary commissions in the Royal Army Medical Corps.

It will be apparent that one absolute standard of vision cannot be rigidly insisted upon for all units. Some allowance must be made for the class of work that the soldier will undertake, and this is considered in other physical tests apart from vision. I have not been able to trace the standard of vision in the British Army further back than 1906, and it is unnecessary to do so, for the standard given at that date was in force up to and during the earlier weeks of the war. It was the following :

(*a*) A recruit had to read $\frac{6}{24}$ with each eye separately without glasses, or, as an alternative—

(*b*) $\frac{6}{6}$ with one eye without glasses and $\frac{6}{36}$ with the other without glasses.

This was the standard for all units of the service, including Militia, Yeomanry, and Volunteers, with the exception of the Corps of Army Schoolmasters, for which vision with or without glasses had to be "good." The use of the word "Volunteers" appears to suggest that

this standard was in existence before the Territorial Force was organised.

In the Regulations amended up to September 30th, 1914, there were three standards, the first or highest of which remained as before, viz. :

(*a*) $\frac{6}{24}$ with each eye separately without glasses.

(*b*) $\frac{6}{6}$ with the right eye without glasses and not less than $\frac{6}{60}$ with the left eye without glasses.

(*c*) $\frac{6}{6}$ with the left eye without glasses and not less than $\frac{6}{60}$ with the right eye without glasses.

This standard (*c*) was requisite for the A.S.C., R.A.M.C., A.O.C., and drivers of the R.A. and R.E. (A.O. 218/1912). It was adopted in 1912.

The second standard, viz. R. $\frac{6}{6}$, L. $\frac{6}{60}$ was subsequently amended to R. $\frac{6}{24}$, L. $\frac{6}{60}$ without glasses. The earliest date of the change that I can find is September 30th, 1915, and this standard was enjoined as the minimum for recruits till the A.C.I., 211, of February 4th, 1917. This Instruction may be quoted in full.

Standard of Vision for General Service :

(*a*) If a man's vision is $\frac{6}{24}$ in one eye *without glasses*, and his right eye can be brought up to $\frac{6}{12}$ with glasses, he will be considered fit for category A.

(*b*) If a man has lost one eye, or has completely lost the sight of one eye, he will not be considered fit for category A.

When we examine these standards we have to remember that those of earlier date were applicable to a voluntary army, a small standing army, which might be called upon to carry out operations in remote parts of the globe, and it was essential that the men composing it should be in a high state of physical efficiency. The man who was dependent on his glasses was rendered inefficient the moment his spectacles were broken or lost ; he was almost as ineffective as if he were a casualty, and remained so until he obtained a new pair.

It may be reasonably asked how he could do this in the Sudan, in East Africa, on the Indian Frontier, or in recent

times at Suvla Bay. He was useless as a fighting man if he could not carry on his job (which was to fight) without the aid of so fragile a piece of apparatus as a pair of spectacles.

The standard accordingly was high in comparison with that of other countries whose armies, always conscript armies, it is needless to say, were chiefly maintained for home defence and were rarely out of reach of necessities and appliances.

The first war-time standard of R. $\frac{6}{24}$, L. $\frac{6}{60}$, aroused a considerable amount of hostile criticism on the ground that it was too high. It was pointed out in the medical journals that it was excluding from the Army many men who would be fit for general service with glasses, and it was asked why we in this country did not conform with the practice in other armies of making the test a test with glasses.

The answer has been given in what I have said about ours having been a small voluntary Army in which only the physically fit were accepted, and called upon, up to August, 1914, to operate far from a base. Further, we as a nation or the then Government, did not, up to that date, contemplate or foresee the possibilities of our being engaged in a war almost on our very doorstep.

If we turn to the practice in other armies of allowing tests with glasses, we have to bear in mind that compulsory service demanded a totally different system. In other countries every lad knew that when he reached a certain age he would enter the army whether he liked it or not. He was brought up with this idea in his mind, and it had an immense influence on his mental outlook. He realised that one day he would be caught up in the intricacies of the military machine and that there was no escape.

Another point of importance is that in continental armies which allowed the test with spectacles, recruiting was going on as a matter of routine all the time and in large numbers, and as practically all the youth of the nation had to undergo military training there were few

exemptions. His training, besides, did not subject him to that intense mental strain which actual warfare creates, and induces many a man to break away at all costs, if by no more complex expedient than by smashing his spectacles.

Nothing throws a more significant light on the conditions prevailing in a voluntary as distinguished from a conscript army than the fact that while the continental text-books and journals on military ophthalmology devote a large amount of attention to malingering and describe an infinite number of tests for its detection, our British text-books on the eye have little to say on the subject. Far from this being a reproach it is a matter about which we should be proud, but it warns us that the time has come when we must take thought for the future.

Although the standard of R. $\frac{6}{24}$, L. $\frac{6}{60}$ was repeatedly emphasised by the War Office as the minimum for men fit for general service, it was by no means rigidly observed, as indeed we all know. Thus men with grave visual defects, high degrees of myopia, dense nebulæ, old iritic adhesions, disseminated choroiditis, optic atrophy, retinitis pigmentosa, cataract, aphakia, vitreous opacities—the list is almost an index of diseases of the eye—men with these defects were passed, and this proved that the visual tests could not have been carried out strictly or at all. The standard was not adhered to in thousands of instances through carelessness or hurry to get as many men as possible through the recruiting office, and the blame for this widespread laxity lies at the door not of the War Office but of the medical men, many of them in civil practice, who undertook the examination of recruits and did not insist upon sufficient time being allowed for their work.

Further than this, there were instances of medical officers who on their own responsibility challenged and over-ruled the War Office regulations, and passed as fit for general service high myopes who could be corrected to $\frac{6}{12}$ or better. Most glaring were the instances of

external diseases, such as blepharitis of old standing, and keratitis, which were allowed into the Army, only to be transferred to hospital before the men had discovered how to get into their uniform. One case of mine had been in the Army for 400 days, 300 of which he had spent in hospital. Another had spent 90 out of his 92 days in the Army in the same way.

It should not be too late even now to suggest a remedy for this state of affairs ; it must eventually be considered if conscription is to be a permanent system with us, and it would be well if we kept the question before us now.

The test of R. $\frac{6}{24}$, L. $\frac{6}{60}$ had one practical advantage in that it could be applied rapidly by any intelligent person, and men who failed to pass it could be referred for further examination to an ophthalmic surgeon attached to a recruiting medical board. From a military point of view it was adequate and met the conditions under which it was applied.

In February of this year the Army Council issued the instructions which I have just quoted. There has not been sufficient time to decide whether it has had the effect aimed at, namely, to bring into Category A men with refractive errors who were outside it under the old standard of R. $\frac{6}{24}$, L. $\frac{6}{60}$. In my experience it has not altered matters to any wide extent. That is to say, men who were in Category A under the old standard remained there under the new. Over a period of eleven weeks during which I examined 723 new cases, not all refractions, only forty-five were raised from B to A, and ten went down from A to B. The other refraction cases remained as they were.

Those who went down a category had nebulæ or amblyopia from strabismus in the R., but were able to reach the old standard of R. $\frac{6}{24}$, L. $\frac{6}{60}$, no matter what the refractive error was. My figures show, I venture to think, that the old standard of $\frac{6}{24}$, $\frac{6}{60}$, was good, provided the eyes were sound, and that the men were willing subjects.

The Instruction covers a wide field and reads as if some latitude were allowed to the ophthalmic surgeon. But anomalies may be mentioned. Suppose a man has R. $\frac{3}{60}$, and L. $\frac{6}{9}$. He is fit for Class A if his right eye can be brought up to $\frac{6}{12}$. But is his efficiency much improved? Is he not a better man without glasses and shooting with his left, than with a high minus glass with perhaps a cylinder added for his right eye, and a low minus glass for his left?

There are numbers of men whose ametropia is not symmetrical, and in their case monocular vision is the rule. In civil practice the defect would be dealt with appropriately, but in military practice and at the present time the improvement in efficiency is not striking, and it would be just as practical to accept the man for Class A without a glass for his right eye and to teach him to shoot from his left shoulder. He has been content to go about the world with all but normal vision, but he is lost to Class A if with the most accurate refraction he cannot be improved beyond $\frac{6}{18}$. The time and patience exhausted in attempting to obtain a better result are thrown away.

It is not necessary to burden you with examples of cases which are puzzling, but one small matter of detail may be mentioned. I have a rifle (short magazine, Lee-Enfield, Mark III), with which I test men when their glasses are issued, and the men are asked to read the test-types while aiming at them with the rifle through their spectacles. I have found that when the correction is above plus or minus 3·00 D or even not so much, and more especially when a cylinder is a component, the vision when sighted by the rifle with spectacles, is always two or more lines worse than with the direct gaze. Some men say their glasses are of no use for shooting in the prone position.

The explanation of the failure of spectacles in many cases to improve rifle-sighting is simple. The man does not look through the optical axis of his glass when sighting; he looks obliquely through the upper and inner

segment. He cannot adjust his head to such a position as to give him the use of the optical axis, and as the refractive power of a lens is altered with the distance from its axis, there is distortion. Periscopic glasses would meet the case, but they are not "issuable," and the decentration of a glass for rifle-shooting would be out of the question. A sight fixed to the small of the rifle would be liable to damage, and in some patterns would be in the way of the bolt.

In practice I have to carry out my instructions, but when testing a man with glasses and rifle I explain to him that he has to get his eye as near the centre of the circle of the spectacle frame as possible; but in many cases the vision for shooting is better without than with glasses.

It is always a great disappointment to the keen soldier when he fails at the range, and it might be well to begin his training with rifle-practice so easy that he is encouraged and interested. In myopes a difficult matter to decide is the amount of the correction that should be given, for a glass which makes the target appear sharp may not be the best for showing up foresight and backsight. It is worth pointing out that the best shot in the American Army, an officer, had only $\frac{3}{60}$ with his right eye, corrected with $-2{\cdot}25$ to $\frac{6}{6}$, but it is not stated that a specially decentrated lens was used. The question of shooting with glasses—shooting as a part of military training, that is, and not match-shooting—is full of difficulties and apparent contradictions, and if many men with normal vision make poor shots, and if others with poor vision can pass their musketry, some account must be taken of intelligence, attention and the muscular balance of the arms in bringing the rifle to the shoulder.

While I am on this subject some reference must be made to the use to which soldiers of a certain type put their surgical appliances, such as dentures and spectacles. About a year ago the War Office sent out a letter pointing out that a soldier would not wear glasses unless he was satisfied that he was deriving benefit from them, and the

suggestion was made that glasses below −1·D and +2·D in men under thirty should not be issued. The letter further laid stress on the fact that quite 40 per cent. of the men to whom glasses had been issued never used them. The number on my last prescription of April 28th, 1917, was 60,029 ; and this shows that the issue has been on a fairly large scale. But we must remember that when a man with R. $\frac{6}{24}$, L. $\frac{6}{60}$, is being tested with trial lenses, we are demonstrating to him the fact that he has an optical defect which can be overcome with spectacles, and by providing him with them we are, above all, supplying him with an excuse if he wishes to evade irksome duty.

You perhaps may think what I have said has no practical bearing on the question of visual tests, but in my experience it influences it at every turn. In theory there should be no distinction between military and civilian efficiency, but it is only when the soldier is regarded as a potential fighting or serving man that the distinction comes to light. If a conscript army were permeated by the same high motives that are found in individuals, the distinction would be obliterated. Voluntary National Service brings to the front one type of man, compulsory National Service forces to the front many of a different type and it is with these that we have to deal. It is idle to discuss or even to think of academic solutions of visual problems when the test can be evaded, especially when appliances like trial glasses are employed.

The test, reduced to its basis, depends on the statement of the man being examined, and the statement of the man who examines him. Again and again, when I have satisfied myself that a man's refractive error is consistent with a vision of $\frac{6}{12}$ or better, I have warned him that my report to his medical officer will be founded not upon what *he* sees but upon what *I* see by means of the ophthalmoscopic examination which I make in every instance, and this often has had the effect of putting him on his guard against reporting sick again on account of his eyes.

Although the instructions for examining recruits are much more elaborate than those of any insurance office, the reference to vision is no more extensive than what I have quoted. All that concerns the recruit, is that if he can read $\frac{6}{24}$ with *one* eye and have sight in the other, he is to be passed as fit provided that the vision of the *right* eye can be brought up to $\frac{6}{12}$ with glasses.

Thus there is not the same stress laid on accuracy of vision as there is on other physical conditions, although we would imagine that a grave visual defect would decide the case finally in spite of highly specialised functions in other directions.

The alteration of the test from one without glasses to one with glasses has completely transformed the work of the ophthalmic surgeon. He has become for the most part a refractionist. The older test of R. $\frac{6}{24}$, L. $\frac{6}{60}$, was, as I have said, so simple that anyone could apply it. But if the right eye has now to be brought up with glasses to $\frac{6}{12}$, there are thousands of men who will need to be re-examined, and the question is, by whom? As you know, there are stations throughout the country called Ophthalmic Centres, established for the provision of spectacles for the troops, and in charge of a medical officer experienced in ophthalmology, who has as his orderly a "certified optician." The duty of this orderly is to measure men for frames, to attend to the duplication of prescriptions, to dispatch spectacles, and generally to carry out the directions of the medical officer. But the pressure which the new standard of vision is creating has quadrupled the refractive work of the Centres, and the temptation will arise—indeed, in some instances it has proved irresistible—when the medical officer, overwhelmed with refractive cases, and not too punctilious in his observance of ethical principles or War Office instructions, will hand over much of the work to the orderly, contenting himself merely with signing prescriptions to which he has not given his personal attention. Thus the classification and fitness of a soldier may depend on the result obtained

by an unqualified man, who obviously is not in a position to diagnose the existence of pathological conditions. This, perhaps, is scarcely a state of affairs foreseen by those who so urgently pressed for a visual test with trial glasses, and in my opinion it constitutes a grave menace to ophthalmology. In the future it will be graver still.

It has already been said this new standard of $\frac{6}{24}$ in one eye, with the right eye improved to $\frac{6}{12}$ at least, sanctions the admission to Class A of men who may have a fairly high degree of myopia in the right eye, and the question to be determined is whether it will lead to increased efficiency to put a myope with R. $= \frac{3}{60} - 6$ D. $= \frac{6}{12}$ into Class A *because* his left eye sees at least $\frac{6}{24}$. Bearing in mind what was said about the influence of a high plus or minus glass on sighting with the rifle, you will appreciate the problem. I am aware that many high myopes went to France early in the war, served in the trenches, and returned unscathed, but when trench-life, and particularly night operations, are taken into account, it may reasonably be asked if these men were endangering, not their own lives, so much as the lives of their comrades, by losing touch or missing their way at a critical moment.

It is not for me to suggest an alteration of the present standard, but to ask you to consider *standards* as a basis of classification of men according to their vision. Test-types cannot be dispensed with, but when a refractive error is present, with no active disease, it might be well to take into account the amount of error as shown by the ophthalmoscope or retinoscopy. This admittedly would not help in cases of amblyopia which presented no objective evidence, but a *dioptric standard* might outline some visual classification, and determine whether a man was fit for a fighting or non-combatant unit. After all, the ophthalmoscope is our only weapon when we take arms against the obstinate and reluctant, and these in their hosts will confront us in the immediate future.

New units are being formed of men who have been at the Front, who have been wounded, who have been sub-

jected to the strain which produces shell-shock. These units are being formed of men who have been brought before one Medical Board after another on account of defective vision. The obdurate and the hanger-back are being called up, and of these there are large numbers who have undergone so many visual tests and so many ophthalmoscopic examinations that they know the safest attitude to assume in their aim—as surely it will be in many instances—to baffle the medical officer.

Among these there are men who have been on active service, and have undergone every conceivable test of physical and mental endurance. There are questions to be decided as to whether a condition of the fundus was caused or aggravated by active service, or was in existence at the time of enlistment, when ophthalmoscopic examinations were not made owing to haste to get the men through—examinations which, had they been faithfully recorded at the time, might have saved the country thousands and thousands of pounds for claims based upon visual defects. Had these defects been seriously examined at the recruiting office, they might have been found to be congenital in origin, or due to pathological conditions in existence previous to enlistment, and therefore not incurred through, or aggravated by, military service.

Were time less exacting, I think I am not the only member of this Society who could say much on this subject. What is most to be desired is finality in authority upon those cases in which definite visual disability in the soldier, or unalterable rejection in the recruit, is decided once for all by one inviolable tribunal.

The experience of others in the R.A.M.C. must be similar to my own. Men have been sent to me for an opinion who have appeared before as many as eight Medical Boards, examined by eight specialists, each of whom has written identically the same report. One M.O. may find tubercle bacillus; another may not. One M.O. may find albuminuria; another may not. One M.O. may chance upon a fleeting cardiac derangement, which has

vanished before the next examiner applies his stethoscope. But a man with anterior polar cataract in each, with retinitis pigmentosa, with optic atrophy, with ruptured choroid, with retinal detachment in one eye and traumatic cataract in the other, with posterior synechiæ in each—the catalogue is endless—such a man has a definite, incontrovertible, and, above all, a *visible* lesion, which should relegate him for the rest of his life to such civilian duties as his vision will allow him to perform. He is not for the Army, and, whatever help he may gain by attendance at a civil hospital, the Army is not for him.

I have endeavoured to place before you some conditions with which we are faced in our efforts to discover a standard of vision. There are vast differences between an army on a peace footing and one on a war footing; between the volunteer and the conscript; between war at a remote distance and war at our doors; between a war of movement and a war of entrenchment and siege. These cannot be ignored; but we are concerned in addition with finding out the best means for making the utmost use of our material, and much depends on the grading of a man according to his vision. We need more than one standard, and if we can arrive even at an outline of what these should be, we have done much.

There is urgently demanded the organisation of a branch of military ophthalmology, staffed by men fully trained in military requirements, who should be entrusted with power to pronounce authoritatively and finally upon a man's visual fitness.

In the brief time allotted to me I have attempted to suggest points for discussion, and it has been my endeavour to avoid rather than to stimulate controversy. For this reason I have not dealt with the writings of those who somewhat urgently have insisted upon our immediate adoption of the continental system of visual tests.

I trust they will not take offence if I hint that this all-important question of vision in the Army, like not a few problems that demanded solution on and after August 4th,

1914, has left theory safely dumped leagues behind the firing line, and has brought us face to face with the implacable barbed wire of experience.

Capt. A. W. Ormond thought it should not be accepted that all Capt. Wallace's statements were absolutely true. As an individual who had some responsibility at one of the ophthalmic centres in London, he was able to assert that there was much which could be said on the other side. In his experience, and so far as he knew, there were no military ophthalmic cases seen by orderlies; but there had been an impossibility in getting the War Office to take the slightest notice of the fact that the ophthalmic surgeon had often to see annually 2000 out-patients who had been sent promiscuously. Yet he and his colleagues had never, under the most urgent considerations, obtained the assistance of orderlies, but had got through the work in addition to their ordinary run of in-patients.

Col. Elliot said that at some at least of the ophthalmic centres the numbers of the patients were quite out of proportion to the staff: there were more than a man could be reasonably expected to deal with in an afternoon. Some men had forty fresh cases to see in an afternoon, and no man could possibly do justice to so many in the time. That fact must be taken into account in any criticisms which might be made.

Mr. Bickerton said he had only one remark to make. As far as the standard of vision was concerned, if the Government wanted men they took them, whatever the standard. If they did not want them, they set up a standard of vision. With regard to the way in which the work was done, how could one expect it to be better, seeing that originally there was no organisation and no equipment provided? A man was sent down to a certain district to examine eyes: he found nothing there—no ophthalmoscopes, no glasses, absolutely no facilities for working. The War Office used these so-called standard tests if they liked, if not, they did not consider them. He

understood that in one town a gynæcological surgeon was engaged in doing these tests. If there was anybody to blame in this matter, it was the War Office : that had been so from beginning to end. Ophthalmic surgeons had done the best they could.

Mr. Harrison Butler said the practice he adopted was to put everyone under homatropine, thus saving time and avoiding mistakes.

Mr. McMullen said he had examined many hundreds of cases at the Whitehall recruiting depôt since the introduction of the new standard, and he was certain the result had been that they had been able to place a largely increased number of men into Class A.

Mr. Inglis Pollock said he knew one depôt which was in charge of a school medical officer, who fifteen years ago had been for one year an eye infirmary house surgeon, and another was in charge of a man who had been an extra dispensary assistant and was recently made clinical assistant : while, on the other hand, he knew an ophthalmic surgeon and two assistant surgeons who were acting as registrars and not permitted to look at eye cases.

Dr. G. Mackay said there had certainly been in the country a great deal of dissatisfaction in this matter, but as the subject could not be threshed out or remedied by a hasty discussion, he suggested that the Council of the Society should take the matter into consideration, and see whether steps could not be taken to make recommendations to the War Office. He would like to propose—

"That the Council be, and is hereby requested, to consider and report to the Society on the conditions affecting the standards of vision in the British Army with the ultimate intention of submitting recommendations to the War Office for the more efficient control of the ophthalmic department of the Medical Service."

Mr. Bickerton seconded, and it was carried.

Capt. Wallace, in reply, said that, in reference to his statement that orderlies were doing the work, it was his duty to inspect a number of ophthalmic centres, and he

knew what was going on. He had seen dozens of prescriptions signed by orderlies with the responsible officer's name: the medical officer had not even taken the trouble to write his own name on the slips.

5. *Remarks upon advancement operations.*

By C. Worth.

Though the advancement operation which I have used for more than twenty years is the same essentially as when it was first described, many little improvements in detail have been made from time to time. All these have been described, save one, of which I will speak later. Though I consider this operation to be now technically perfect, of course one does not suggest that it is not possible to produce good results by other advancement operations. A painter needs the best pigments procurable, but can get on well with a lower quality, for these go a very small way towards the making of a picture. The skill and experience of an operator are of vastly more importance than the method of operating or the materials which he uses. But there are certain facts which should be carefully considered by everyone who undertakes an operation for squint.

(1) If the method of attaching the sutures to the muscle or to the globe allows the sutures to cut through or to give ever so little an accurate result cannot be expected.

(2) Very great care is needed to place the sutures symmetrically and to equalise the tension in tying them, in order to avoid elevation or depression of the globe or rotation about its fore-and-aft axis. For this reason any method of fixing the sutures above and below the globe is inadmissible.

(3) Tenotomy of the opponent muscle (apart from the danger of the eye ultimately turning the other way), being

uncertain in its immediate results, renders any approach to accuracy impossible.

(4) Shortening a muscle and re-attaching it to its old insertion, while suitable for very small deviations, cannot be depended upon to produce any considerable effect, and the attempt usually results in a diminution of the range of rotation of the eye in both directions.

(5) Simple shortening of a muscle with tenotomy of its opponent appears to involve a greater risk of ultimate divergence than advancement and tenotomy.

(6) In advancing a muscle it is of great importance as far as possible to avoid disturbing its relations, and especially to avoid abrading its under-surface. A so-called advancement operation in which the muscle is first separated from surrounding structures at best becomes a mere shortening, for the underside of the muscle forms adhesions to its old insertion. In some cases in which this had apparently been done, without producing the desired result, I have subsequently been called upon to operate upon the same muscle. The adherent mat of muscle and scar tissue was found difficult to deal with.

The operation which I use is a true advancement; adhesions are not formed between the under-surface of the muscle and its old insertion. This I have proved when operating a second time upon the same muscle in some cases in which I had produced too large or too small a result.

The improvement of which I spoke is in the use of a special scleral needle. In my earlier operations the sutures not infrequently cut their way out of the sclera before the end of the muscle had become firmly united to what was intended to be its new insertion. Gradually I put the sutures deeper and deeper into the sclera until a depth was found at which they never cut out and which has proved to be safe. For many years I used just this depth without knowing precisely what proportion of the thickness of the sclera it represented. Three or four years ago my sutures were inserted in an eye which was about

to be excised for sarcoma, and Mr. Greeves very kindly cut sections for me. The sutures penetrate three-fifths of the thickness of the sclera. A shallower position cannot always be depended upon; there is not much room to go deeper within the limits of safety. I have never had an accident myself, probably because this depth was arrived at very gradually. But I have seen in consultation a case in which the eye was destroyed owing to sutures reaching the interior of the eyeball, and one has heard of other cases.

Accurate insertion of these sutures is rendered unnecessarily difficult by the large expenditure of force required to make an ordinary eye-needle traverse about an eighth of an inch of scleral substance. The sclera may easily be cut with a sharp instrument; it may be torn with great difficulty, but it cannot be stretched to any appreciable extent. A needle, sharp at the point but not at the edges, can only pass through scleral substance by a tearing action like that of a wedge. One knew what the needle should be, but for a long time it proved impossible to get an instrument-maker to realise that one wanted a very perfect surgical instrument, not drapers' wares put up in paper packets and sold by the dozen. At last Messrs. Weiss made them for me. The needle is straight, three-quarters of an inch long, and has a split eye. The shaft is round. The head is trowel-shaped, each edge being as sharp and as carefully finished as the edge of a Graefe knife. The head is of such a width as to cut a slit in the sclera through which the round shaft of the needle and the suture will just pass easily. The needle is straight, because the instrument-maker has found it impossible to make perfect edges to a curved needle, owing to the difficulty in holding it. The split eye is to enable one to use curved needles for the muscle. A straight needle cannot well be used on the lateral recti muscles of a deeply-set eye nor upon the inferior or superior recti in any eye. The needles are kept in a wooden case made to hold them separately, so that the heads do not touch anything,

and they are sterilised in absolute alcohol, so that no one need touch them but oneself.

For inserting the sutures in the muscle, small curved eye needles are used. Then, as each suture is to be inserted in the sclerotic, the curved needle is removed and the suture slipped into the split eye of the scleral needle. One must remember to keep the trowel-shaped head of this needle parallel with the surface of the sclerotic.

This needle is a help in an operation, the *accurate* performance of which requires more judgment and experience than any other operation in ophthalmic surgery. In a squint operation (given adequate nursing, which one may always command in private practice) the result depends entirely upon the surgeon, who should accept responsibility for it.

Mr. Arnold Lawson asked whether Mr. Worth handled the thread in order to pass it into the needle. He asked the question because he thought there was an objection to touching thread, for operation, with one's fingers, and he personally took the greatest care not to handle either needle or thread at any period of the operation, using forceps and needle-holder alone for every purpose.

Mr. Harrison Butler said he wished to ask, in all humility, how Mr. Worth managed to split the sclera into fifths. The sclera, at the point mentioned, was ·8 mm. in thickness, and to go through three-fifths of it seemed an extraordinarily difficult thing to manage.

He had found it difficult to get results with pure advancement without tenotomy. In the vast majority of cases, whatever one did, the stitches seemed to have cut out of the sclera by the pull exerted by the untenotomised muscle. He found one could do a tenotomy, not letting go of the muscle, and suturing through it to keep it in position. He did not lay the muscle back, but paralysed it for a few days, so that it might re-attach itself to its old position. He did not see why there should be any objection to a muscle forming an attachment by its belly

to the old position. His anchor-suture method aimed at that, and since he had adopted that, his results had been better than before.

Mr. J. Gray Clegg said that in one or two cases in which there had been a marked convergence, in which he had done advancement without tenotomy, the eye had been pulled back into the orbit, and the palpebral fissure was distinctly less than on the other side. It was sufficiently conspicuous to be noticed by any ordinary person. In such cases tenotomy of the opposing muscle, combined with the advancement, gave a better cosmetic result.

Mr. Claud Worth, in reply, said Mr. Harrison Butler wanted to know how he, the speaker, could tell he was in three-fifths of the thickness of the cornea. He used the method for years without knowing in the least. At first he found that his sutures sometimes cut out, and gradually and slowly he got deeper and deeper. When he at length got to the depth at which they held, he kept to that depth. It was not until three or four years ago that he had the curiosity to find out what proportion of the thickness of the sclera he had gone through. Then he found it was three-fifths. He wondered whether any member knew what effect was going to be produced by tenotomising, and then advancing the muscle on the other side. That tenotomised muscle had to be re-attached afterwards, and no one could foresee what the effect would ultimately be. If, however, one were doing an advancement alone, the ultimate result of the operation would depend entirely upon the surgeon.

With regard to attaching the belly of the muscle to the old attachment, that would not yield such good results, because the power and range of action of the muscle would then be not so good. But if a surgeon had doubts about being able to get the end firmly adherent to the sclera, he had better sew it all the way down to its old insertion than do a tenotomy on the other side.

In reply to Mr. Gray Clegg, if one produced a large

effect by advancement alone, it caused retraction, he agreed; but for anything over 15° in cases of convergent squint, he advanced both external recti, but not both at the same time. He divided the effect between the two eyes.

In answer to Mr. Brooksbank James, before he, Mr. Worth, got Weiss to make the needle he now worked with, he experimented with Mr. James's needle. An objection to it was, that the hole it made was not sufficient to allow the thread he used to pass through, and hence he had to use a finer thread than he would like to. Also he found the needle tôo flexible, and it had not a sufficiently good cutting edge.

With regard to putting two sutures transversely to the axis of the muscle, he did not think there was any objection to that, provided one got it all symmetrical. There were many different methods by which one might produce good results, so long as one always used the same operation and got thoroughly accustomed to it, and gradually, by experience, learned what was likely to happen to the case aftewards.

6. *Functional spasm of accommodation.*

By Leslie Paton.

True spasm of accommodation is one of the rarer conditions met with in ophthalmology. Hirschberg, in 1884, said: "I scarcely believe in the existence of acute and not at all in the existence of a chronic form of accommodative spasm. I have never seen one case out of many thousands tested by the ophthalmoscope and by glasses." Hess, in the second edition of Græfe's *Sæmisch*, also expressed a similar feeling as to the rarity of true spasm of accommodation. In our own *Transactions* I have been able to find only two papers

dealing with the subject; one by Adams, in vol. ii, p. 180; and one by Fitzgerald, in vol. iv, p. 311.

Adams gives two cases; the first, a girl, æt. 13 years, who had had diphtheria in September, 1880. In December, 1880, her visual acuity with + 2 D. sph. in each eye was $\frac{6}{6}$. In August, 1881, he found she required R.E. − 5 D. sph. = − 1 D. cyl. ax. hor. to get $\frac{6}{6}$, and L.E. − 4 D. sph. to get $\frac{6}{9}$. After using atropine the refraction was found to be practically as it had been in December—

R.E. + 2·5 D. sph. = + ·5 D. cyl. ax. V. = $\frac{6}{6}$.

L.E. + 2 D. sph. = $\frac{6}{6}$.

His second case was by no means as striking, and I will not quote it.

Fitzgerald also gives two cases. In the first, a myopia in one of − 3 D. disappeared completely under atropine. The other eye showed no spasm, and was emmetropic. In his second case, a myopia of − 2·25 D. in each eye proved, under atropine, to be a hypermetropia of R.E. + 1·25 D. sph., and L.E. + ·75 D. sph.

Before proceeding further I must guard myself by defining what is meant by true spasm of accommodation, and I cannot do better than paraphrase the words of Hess. Much misunderstanding has arisen from the want of a sufficiently sharp limitation of what is meant by so-called spasm of accommodation. It is not right to call the condition of maintained accommodation found in young hypermetropes a ciliary spasm: it is really a physiological adaptation in the interests of clear vision. A slight degree of true spasm of accommodation is frequently found, especially in myopic school children. Here there can be no question of its subserving the interests of clear vision, and there can be little doubt that some degree of spasm of the ciliary muscle exists in a very definite percentage of myopes, and even of school children in general. Stocker found it in 5 per cent. of cases investigated, and Schmidt Rimpler in 10–30 per cent; while the proportion in myopes was even higher. The difference produced in such cases as a rule by the

use of full atropisation is not more than 1 D. to 2 D. The development of this more or less constant contraction of the ciliary muscle is not, however, a spasm of accommodation in the sense in which I use the term to-day. It may only be a phenomenon associated with the increased convergence that uncorrected myopes and astigmatics adopt to get clearer vision. If so, it belongs to a category analogous to the class of hypermetropes spoken of above. Apart from these two types, in which some contraction of the ciliary muscles exists, true spasm of accommodation must be regarded as a comparatively rare occurrence. I would define it as the sudden development in one or both eyes of a considerable degree of apparent myopia, which disappears when the eye is atropised. The spasm may be continuous or clonic, or may pass from clonic to continuous, and it may be associated with spasm in other ocular muscles.

Case 1.—*History.*—Miss M. M— is an extremely intelligent and very healthy young lady. I saw her first in May, 1909, when she was 13, but I afterwards received some notes from the late Dr. Fitzgerald, in Dublin, which enable me to give her history from the age of 5. He writes: "I first saw the child in 1907. She was then 5 years old, and she was brought to me because her left eye was observed to *converge*. V. = $\frac{6}{18}$ E.E. Retinoscopy + 1·5 D. E.E. I saw her again in 1903 V. = $\frac{6}{9}$ E.E. I prescribed for reading + ·75 D. sph. E.E. She came to me again in 1907, as she had lost her glasses. R.E. V = $\frac{6}{6}$, L.E. V. = $\frac{6}{6}$. Left eye had again been observed to converge. I prescribed + 1 D. sph. E.E. Some time after this they went to London, and I did not see her again till she came to see me last February with your letter and notes. In 1884 I reported two cases of 'acute spasm of the accommodation' (see reference above). I took a great deal of trouble at the time in searching up all the cases on record, and could lay my hands on few. As well as I can remember, there were a couple reported in *Græfe's Archives*, but they were both connected with

some trauma of the cornea. If I am not mistaken, Donders expressed some scepticism of the existence of such a condition." I have quoted a large part of Dr. Fitzgerald's letter because of its general interest, but as regards Miss M. M— the important fact is that at æt. 5 years there was *convergence*, occasional but definite, of the left eye. When I saw her in 1909, at the age of 13, the history was one of a tendency to divergent strabismus when tired. I found the R.E. emmetropic and the L. E. — ·5 D. myopia. There was quite *definite divergence on screening*, but good stereoscopic vision, and the eyes were quite straight when both were uncovered.

Under atropin R.E. refract. = + ·75 D. sph., L.E.= — ·25 D. sph.

In December, 1910, with no mydriatic R.E. was emm. V.A. = $\frac{6}{9}$.

L.E. c. — 1·5 D sph. ⌒ — ·5 D. cyl. ax. 10° V = $\frac{6}{9}$.

There was still a marked tendency to divergence.

I ordered a correcting glass for the left eye and stereoscopic and convergence exercises. In April, 1911, her visual acuity was $\frac{6}{5}$ in each eye (with correction in the left eye). She had good stereoscopic vision and the divergence was much less frequent. No change of any importance was noticed till the end of 1912, when, for the first time, she complained of seeing double when she did not use her glasses. She still got good stereoscopic vision with her glasses and $\frac{6}{5}$ E.E. The complaints of seeing double increased, especially on reading, and by April, 1913, a permanent divergence of 14° of the left eye had established itself. Under observation this divergence remained steady at 14°, so I suggested the advisability of doing a tenotomy of the left external rectus. This was done, with the result that at the beginning of June her eyes were quite straight. She could see things without doubling for much longer periods, and when she got diplopia it was *homonymous*. Her refraction tested at the end of June without atropine was R.E. — ·75 D. sph. ⌒ — ·25 D. cyl. ax. V = $\frac{6}{6}$. L.E. $\frac{\text{— 2 D. sph.}}{\text{— ·5 D. cyl. ax. 10°}}$ = $\frac{6}{6}$.

By September, 1913, a very variable convergence easily shown by screening or by the Maddox rod had manifested itself. Her eyes still looked straight with glasses, but the homonymous diplopia was much more troublesome, and with the Maddox rod 15° to 20° of prism was required to overcome the diplopia. Mr. Holmes Spicer saw her with me at that time and advised against further operation and suggested the use of an opaque glass in front of the left eye to avoid the worrying diplopia.

On October 27th, 1913, she appeared complaining of very bad vision. There was now quite manifest convergence variable in amount from 10° to 14°, but the most remarkable feature was an apparent myopia of — 7 D in the right eye and — 9 D. in the left eye. This was variable and seemed to increase and diminish with the variations in the convergence. Under full atropine mydriasis her refraction was R.E. + ·5 D. sph. = $\frac{6}{6}$. L.E. — 1 D. sph ⌒ — ·75 D. cyl. ax. hor. = $\frac{6}{6}$. I ordered her R.E. + ·25 D. sph., L.E. — 1·25 D. sph. ⌒ — ·75 D. cyl. ax. hor., and on testing her with these a few days later she could get the images together with 9° prm. base out. The spasm recommenced as soon as any attempt to abandon the use of atropine was made. She could get her eyes straight, but even then the diplopia still persisted.

In January, 1914, Mr. Lawford saw her along with me and we made a very careful examination. When first examined she showed a spasm of right accommodation requiring — 6 D. sph. to get $\frac{6}{9}$ vision. By gradual reduction of the strength of the glass we got her to see $\frac{6}{9}$ with — ·25 D sph. Then she had an alternating spasm with a period of about 7 secs. While this was happening there also was a definite variability in the amount of convergence of the left eye and also in the size of the palpebral opening.

At this time she was sent off to Ireland to have a holiday and incidentally Fitzgerald re-examined her. He reported in May, 1914, that while under his obser-

vation no change had taken place. "The spasm of accommodation reappeared in the right eye just as acutely on giving up the use of the drops. The angle of convergence in the right eye as measured by the perimeter was 27°. In making this test I noticed a curious nystagmus-like movement of the right eye for some seconds before it became fixed."

On the suggestion of Fitzgerald we sent Miss M— over to Paris to get Landolt's opinion, which I must quote nearly in full.

"I have examined Miss M. M— on April 30th and May 1st and 4th of this year (1914). I have found her the subject of a spasm of accommodation, which, in the absence of any mydriatic, changes the refraction of the eyes about 9 D. From a myopia of 1 D. in the left it increases to 10 D., and from a slight hypermetropia in the right it changes to — 9 D.

"Miss M— has besides a very marked convergent strabismus (approximately 27°). She uses the left eye for fixing and the right eye is deviated towards the nose, but not always to the same degree. This eye, on the contrary, shows definite spastic contractions, almost nystagmic, which tend sometimes to turn it into an extreme degree and then momentarily relax. The convergent strabismus of Miss M— is in part a *concomitant* strabismus due to the spasm of convergence which accompanies that of accommodation. But it is due also to the *tenotomy of the left external rectus muscle.* In effect, as I have shown, the *tenotomy of an oculo-motor muscle is equivalent to a paresis.*

"The convergent concomitant strabismus is then in this case complicated by a paretic strabismus. That is shown, amongst other signs, by the limitation of the temporal movement of the left eye, much more marked than that of the other eye, which is not beyond what is usually seen in concomitant convergent strabismus.

"So, as the patient prefers to use the left eye for fixing, that is, the paretic eye, the paretic deviation shows itself

in the other or right eye. The deviation is thus greater than when the right eye fixes, because it is a *secondary deviation.* It is in this way that we get the explanation of the spastic movements of the right eye, of which I have spoken above.

"As to treatment, I consider that it is above all necessary to advance the external rectus muscle of the left eye, while continuing energetically and regularly the treatment of the accommodative spasm by means of mydriatics.

"It is more than probable that this operation will not be enough to correct the strabismus, but that it may be necessary to advance also the right external rectus. But that operation can be reserved for a later period, when the effect of the advancement of the left external rectus can be considered definite; that is to say, about one month after the first operation."

Such was the report that Miss M— brought back with her from Paris, and with most of it one could not help finding oneself in agreement. Unfortunately its lucidity and straightforwardness has been attained to a certain extent by omitting to take into account some of the complexities of the case.

Here is a girl who at the age of 5 has an occasional convergent strabismus which has turned into an occasional divergence by the age of 13; at the age of 13 she has perfect stereoscopic vision, and yet at the age of 16 she begins to develop occasional diplopia, and by the age of 17 has developed a constant divergence of 14°. This divergence is seemingly cured by operation, but the diplopia remains though it changes its character. Then suddenly an acute accommodative spasm develops and at the same time a convergence spasm.

I felt at the time that Dr. Landolt's note did not help us much to get at an explanation of the complexities of the case, and I think I may say that Mr. Lawford, Mr. Spicer, and myself were of one opinion as to the inadvisability of any further operative interference in the conditions then existing.

When I saw her after her return from Paris I found the variability of convergence much greater than on any previous occasion. On fixing with the left eye, which she still preferred to use, the angle of convergence varied from 18° to 32°. On using the right eye for fixation the angle of convergence occasionally reached 36°, but on the whole the movements were very similar. This observation was not in agreement with one of Landolt's, who found the deviation greater when fixing with the left eye, and as he based part of his argument on the existence of a paretic secondary deviation of right eye when the left eye fixed, I repeated it carefully and confirmed the fact that, as a rule, though there was no great difference between the angles observed on fixing with either eye, on the whole they were slightly greater on fixing with the right eye. It was also noted that there was a periodic spasmodic opening of the palpebral apertures of the two eyes.

Miss M—'s father is a doctor and he considered that, having got Landolt's opinion so strongly in favour of restoring the tenotomised left external rectus to its original position, this should at least be tried. It was against my own judgment and against Mr. Lawford's, but Dr. M— was willing to accept the responsibility. I felt sure that under an anæsthetic all signs of the spasm would go, and I asked the father to be present and see what took place then before deciding finally. As soon as she came fully under the influence of the anæsthetic it turned out as I expected. The visual axes were parallel, if not slightly divergent. However, Dr. M— decided that he would prefer me to go on with the operation. After the advancement the left eye was divergent to an angle of between 15° and 20°. Both eyes were kept bandaged up until the stitches were removed on the eighth day. Then it was found that she preferred to use the right eye, and that on fixing with the right eye there was no spasm and the left eye diverged to an angle of about 22°. But as soon as she attempted to fix with the left eye, spasm

commenced developing rapidly up to — 10 D. of myopia, while the right eye wandered wildly from a position of parallelism to one of extreme convergence. The absence of spasm on right eye fixation lasted for one week, from June 2nd to June 9th, when definite spasm in both eyes again appeared: R.E. — 9 D, L.E. — 10 D. Under atropine this disappeared, and on June 15th she showed a hardly noticeable convergence, but had a very definite diplopia which was not affected by prisms in front of the right eye but could be very sensibly diminished by prisms in front of the left eye.

Her refraction under atropine at this time was R.E. + ·5 D. sph. = $\frac{6}{6}$ (2), L.E. — 1·5 D. sph. $\frown$ — ·75 D. ax. 15° = $\frac{6}{6}$ (3), and now she has swung back to a preference for using the left eye, and the visual axes were varying from slight divergence to a convergence of 10° but were never quite steady.

In October and November, 1914, she was kept without atropine for four weeks. The convergence was as variable as ever, and it was noticed that there was a variability in the size of the pupils in a uniform diffuse light. As a general rule they were from 2–3 mm. in diameter, but occasionally they dilated to over 4 mm. The apparent myopia was much as before: R.E. — 8 D., L.E. — 10 D. On January 20th, 1915, the spasm suddenly disappeared and definite divergence with crossed diplopia came on. Her vision was, R.E. $\frac{-\text{·5 D . sph.}}{-\text{·25 D. cyl. 135°}} = \frac{6}{9}$. L.E. — 1 D. sph. $\frown$ — ·75 D. cyl. ax. 15° = $\frac{6}{9}$. In March she was still getting no spasm on using the right eye, but getting it on attempting to use the left eye.

She went on more or less unchanged and using the right eye until February 19th, 1916, when the spasm came on again quite suddenly, and the divergence of about 15°, which had been present for over a year, was replaced by definite spasmodic variable convergence. A few days' treatment with atropine abolished the spasm, but she again reverted to the use of the left eye, and on forcing

the attempt to fix with the right eye, spasmodic convergence at once commenced. On March 15th, 1916, the right eye was divergent to an angle of 31°, and on attempting to get her to use her right eye the myopia of the left rapidly changed from — 2 D. sph. c̄ cyl. to — 9 D. sph.

On April 3rd, after atropine had been used steadily in the right eye only, she commenced using that eye, and the left eye showed — 10 D. myopia.

By May 4th the spasm had gone from the left eye and the use of drops was stopped, and she found that she again was using the left eye, and on attempting to use the right eye spasm developed in both, which persisted for some seconds after she was allowed to revert to the use of the left eye.

Since then she has used only the left eye, and has shown a fairly steady angle of divergence in the right eye, with a considerable spasm of accommodation in it. I have had to increase her myopic correction in the left eye, as she is determined not to allow her peculiar ocular conditions to interfere with her work at the Ministry of Munitions.

And so she remains at the present time, a problem which I may name "functional spasm of oculo-motor muscles, especially of the ciliary muscles," though naming it does not explain it.

I feel that it must be in some way allied to the rare cases, one of which Grieve showed some few years ago at a meeting of the Section, where there was a spasmodic rhythmical contraction of the pupil.

Case 2.—You will be glad to know that this case I can refer to very briefly, and I have little doubt that other members have seen cases like it since the war began.

Pte. C. J— had concussion on February 14th, 1915, the result of a blow on the head on a wooden platform. When I saw him first on July 17th, 1915, he had marked spasm of convergence, blepharospasm, and great limitation of his visual fields. He was then under atropine, and his

refraction, as well as the blepharospasm, would allow me to estimate it was + 1 D. sph. in each eye. I stopped his mydriatic for a few days, and then found that he had — 6 D. sph. of myopia in each eye. The further history in brief was that the blepharospasm and convergence spasm steadily diminished without much alteration in the accommodation spasm, which was still definite in October, when I finally gave up the use of atropine.

On January 29th, 1916, I found his vision was $\frac{6}{18}$ each eye, and with — ·5 D. cyl. in each eye $\frac{6}{6}$. On February 19th all trace of spasm had disappeared. His fields were full, and his vision $\frac{6}{6}$ without any correction.

He was discharged from the Army, and is, I believe, employed in munition making, but I have recently heard that he has developed some form of fits. I have tried to get him to come up for examination to see whether these are functional or organic, but I have not yet seen him.

This second case is in a way much more straightforward than the first one, and probably many members have seen allied cases. Certainly functional blepharospasm has been of fairly frequent occurrence. Often this has been too marked to allow of the other phenomena being investigated. In a very definite proportion of these cases careful inquiry shows a neuropathic history either in the individual or the family. In my first case there is no indication of any other neuropathic tendency, and the condition would seem to be due to an extremely localised functional disturbance of the third nerve nuclei present in a modified form from early life, but excited into much greater activity by the tenotomy done in May, 1913.

XIV. APPENDIX AND MUSEUM SPECIMENS.

Appendix.

The following cases were also shown:

1. Equatorial staphyloma of an unknown ætiology. By J. B. Lawford.

2. Essential shrinking of the conjunctiva. By H. Grimsdale.

3. Congenital cataract, heterochromia, and persistent hyaloid artery. By J. H. Parsons.

4. Aniridia. By J. H. Parsons.

5. Neuro-fibromatosis of the eyelids and orbit, with congenital glaucoma on the same side. By A. C. Hudson.

6. Choroidal degeneration. By R. A. Greeves.

7. A mass at the right macula, ? tubercular. By R. A. Greeves.

8. Nodular elevations on the surface of the iris, which has been subject to a very chronic irido-cyclitis. By R. A. Greeves.

Museum.

Under the care of W. H. McMullen.

Ophthalmoscopes were lent for exhibition by—

Sir Anderson Critchett, Bart., Messrs. Treacher Collins, Priestley Smith, Leslie Buchanan, Adams Frost, H. E. Juler, J. B. Lawford, W. Wallace, Mayou, and Prof. W. D. Halliburton, The Executors of the late Mr. W. H. Jessop.

Messrs. John Trotter, J. H. Sutcliffe, Curry & Paxton, J. Weiss & Son, and C. W. Dixey & Son.

Lt.-Col. R. H. Elliot exhibited a collection of eyes showing cataracts couched by native Indian operators, also enlarged stereoscopic photographs of many of the specimens. (This collection had been presented to the Royal College of Surgeons, and was lent for exhibition by courtesy of Prof. A. Keith.)

Other exhibits:

Exostosis removed from the ethmoid. By J. A. Craig.

Sight-testing obturator. By A. R. Galloway.

REPORT OF COUNCIL.

The Council is happy to report the continued prosperity of the Society.

During the session 1916–17, 8 new members have been elected, and 1 has been re-elected.

On the other hand, the Council has to deplore the loss of 6 members of the Society by death. By the untimely death of its President, Mr. Walter H. Jessop, the Society and British Ophthalmology have suffered a grievous loss. Mr. R. W. Doyne was Vice-President of the Society from 1904 to 1907. Dr. P. W. Maxwell was Vice-President from 1912 to 1915. Lieut.-Col. Ernest Hudson, I.M.S., has also died. These were three of our oldest members, while the small group of original members is further diminished by the death of Dr. K. A. Grossmann, of Liverpool. Capt. John Cropper, R.A.M.C., of Chepstow, went down in H.M. hospital ship 'Britannic,' lost in the Ægean Sea in November, 1916.

The Society now has 499 Ordinary Members and 7 Honorary Members.

In view of the vacancy in the Presidential office occasioned by the death of Mr. Jessop, the Council, acting under the rules of the Society, elected Mr. Treacher Collins as President.

The Fourth Annual Congress of the Society was held on May 4, 5, and 6, 1916, and was in every way successful. Thanks are due to the Committee of Management of the Central London Ophthalmic Hospital in connection with the holding of the Clinical Meeting at their Institution, to the Honorary Staff of the Hospital for the excellent arrangements for the comfort and convenience of

members; and to the authorities of the Royal Society of Medicine for the efficient organisation of the meetings held in their rooms.

The Honorary Treasurer's report and statement of accounts are submitted. From this statement it will be seen that there is an adverse balance of £15 7s. 5d. On this account, and in order to maintain the present high standard of the *Transactions*, the Council recommends that the annual subscription should be raised to One Guinea.

The Council has had under consideration a scheme whereby other Ophthalmological Societies throughout the British Empire may enter into affiliation with our Society. The Council approves of the scheme, and the alterations in the rules required to carry it out will be submitted to the Society.

ACCOUNT OF THE RECEIPTS AND PAYMENTS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM, FOR THE YEAR ENDED APRIL, 1917.

Receipts.

1916–17.	£ s. d.	£ s. d.
To Balance from last account		19 2 2
,, Subscriptions:		
Through Bank	77 18 6	
Through Collector	120 16 6	
	198 15 0	
Less returned Subscriptions	1 5 6	
		197 9 6
,, Dividends:		
East India Railway Stock	14 12 8	
Ramsgate Corporation	11 9 6	
		26 2 2
,, Nettleship Prize Fund		6 15 0
,, Sale of 'Transactions'		30 14 0
,, Adverse balance		15 7 5
		£295 10 3

Payments.

1916–17.	£ s. d.	£ s. d.
By Printing 'Transactions' and other Printing	274 5 6	
,, Less Contribution for Illustrations	10 0 0	
		264 5 6
,, Royal Society of Medicine		5 3 0
,, Reporting		14 1 7
,, Collector		11 16 0
,, Bankers' Commission		0 4 2
		£295 10 3

April 16th, 1917.

Examined and found correct,

J. Cole Marshall,
R. Rutson James, } *Auditors.*

INDEX.

PAGE

Abnormalities, congenital 166
Accommodation, functional spasm of (Leslie Paton) . . 370
Advancement operations, remarks upon (C. Worth) . . 365
Aniridia (J. H. Parsons) 381
Army, conditions affecting standards of vision in an (W. Wallace) 349

Balance-sheet 386
Berry (Sir George A.), Bowman Lecture: on colour-sense phenomena and some inferences which they seem to suggest 5
——— discussion in cases of Leber's disease . . 261
——— discussion on concussion injuries of the visual apparatus 126
——— ——— protection of the eyes in warfare . . 183
——— ——— the value of the ophthalmometer in estimating the refraction of the eye . . . 347, 348
Buchanan (Leslie), unusual case of opaque nerve-fibres . 166
Bickerton (T. H.), discussion on conditions affecting standards of vision in an army 363
Blepharospasm, functional, in an old man (I. L. Tuckett) . 188
Blind, employment of the, discussion on the, introduced by Sir Arthur Pearson, 133; Dr. W. C. Rockliffe, 139; Mr. Arnold Lawson, 144; Mr. G. H. Grimsdale, 150; Capt. A. W. Ormond, 157; Mr. J. Rowan, 159; Mr. Treacher Collins, 159; Capt. R. R. Cruise, 160; Sir Arthur Pearson's reply, 161.
Bowman Lecture, colour-sense phenomena and some inferences which they seem to suggest (Sir George A. Berry) . 5
——— rules of xxxviii
Bowman Lecturers x
Buphthalmos, congenital (H. Grimsdale) . . . 175

PAGE

Butler (Harrison), discussion on advancement operations . 368
——— ——— concussion injuries of the visual apparatus . 128
——— ——— conditions affecting standards of vision in an army 364
——— ——— detachment of the retina at the ora serrata . 237
——— ——— protection of the eyes in warfare . . 183

Cataract, congenital, heterochromia and persistent hyaloid artery (J. H. Parsons) 381
——— history of couching for (Lt.-Col. R. H. Elliot) . 264
Choroid, leuco-sarcoma of, and epithelioma of lip (J. A. Valentine) 229
Choroidal degeneration (R. A. Greeves) . . . 381
Ciliary nerves, herpes zoster affecting the (M. S. Mayou) . 226
Clarke (Ernest), value of the ophthalmometer in estimating the refraction of the eye 343
Clegg (J. Gray), discussion on advancement operations . 369
——— ——— congenital buphthalmos . . . 175
——— ——— pulsating exophthalmos . . . 186
——— ——— treatment of Mooren's ulcer . . 207
——— ——— sclero-corneal trephining for hypertension . 308
Collins (E. Treacher), discussion on case of opaque nerve-fibres 170
——— ——— employment of the blind . . . 159
——— ——— hereditary glioma of the retina . . 246
——— ——— pulsating exophthalmos . . . 187
——— ——— treatment of Mooren's ulcer . . 206
——— ——— unilateral melanosis of a segment of the iris . 174
——— pathological report on Mr. Lawson's case of detachment of the retina at the ora serrata . . . 234
——— Presidential address 1
——— sequelæ of hypotony 281
——— small, tortuous veins on the optic disc . . 171
——— subchoroidal hæmorrhage from concussion, and its relation to—(*a*) "commotio retinæ"; (*b*) "holes at the macula"; (*c*) "pigmentation of the retina"; (*d*) "pigmentation disturbances at the macula" 112
(?) Colloid nodules in the choroid in a girl (M. S. Mayou) . 227
Colour-sense phenomena and some inferences which they seem to suggest (Bowman Lecture, Sir George A. Berry) . 5
Commotio retinæ, relation of subchoroidal hæmorrhage from concussion to (E. Treacher Collins) . . . 112

PAGE

Concussion injuries of the visual apparatus, discussion on, introduced by Capt. A. W. Ormond, 60; Dr. S. A. Kinnier Wilson, 92; E. Treacher Collins, 112; Capt. R. R. Cruise, 122; Sir George Berry, 126; Mr. A. L. Whitehead, 126; Dr. G. Mackay, 128; Mr. Harrison Butler, 128; Mr. B. Cridland, 129; Mr. N. Ridley, 129; Capt. Ormond's reply, 130.

——— subchoroidal, hæmorrhage from (E. Treacher Collins) 112

Congenital abnormalities 166

——— buphthalmos (H. Grimsdale) . . . 175

Conjunctiva, essential shrinking of the (H. Grimsdale) . 381

——— papilloma of the (M. L. Hepburn) . . . 190

Cornea, diseases of the 205

Craig (J. A.), exostosis removed from the ethmoid . . 382

Cresswell (F. P. S. C.), discussion on the value of the ophthalmometer in estimating the refraction of the eye . 347

Cridland (Bernard), discussion on concussion injuries of the visual apparatus 129

Critchell (Sir Anderson) discussion on the sequelæ of hypotony 301

Cruise (Capt. R. R.), discussion on concussion injuries of the visual apparatus 122

——— ——— employment of the blind . . . 160

——— ——— hereditary glioma of the retina . . 247

——— ——— protection of the eyes in warfare . . 176

Couching for cataract, history of (Lt.-Col. R. H. Elliot) . . 264

Council report 383

Davies (D. L.), iridocyclitis of obscure origin . . 220

Elliot (Lt.-Col. R. H.), discussion on conditions affecting standards of vision in an army . . . 363

——— ——— protection of the eye in warfare . . 183

——— ——— pulsating exophthalmos . . . 186

——— ——— sclero-corneal trephining for hypertension . 321

——— ——— small tortuous veins on the optic disc . 173

——— ——— the sequelæ of hypotony . . . 303

——— ——— history of couching for cataract . . 264

Equatorial staphyloma of an unknown ætiology (J. B. Lawford) 381

Evans (J. Jameson), fundus lesions in cases of war nephritis . 238

Evolution of the ophthalmoscope (W. H. McMullen) . . 324

——— of visual perceptions, apocritic principle of, the (J. Herbert Parsons) 333

PAGE

Exophthalmos, pulsating, as the result of a wound of the head (M. S. Mayou) 186
Exostosis removed from the ethmoid (J. A. Craig) . . 382
Eyelid and orbit, diseases of the 188

Fisher (J. Herbert), Leber's disease, and two allied cases associated with changes in the sella turcica . . 251
——— two cases allied to Leber's disease, but without family tendency 256
Functional spasm of accommodation (Leslie Paton) . . 370
Fundus lesions in cases of war nephritis (J. Jameson Evans) . 238

Galloway (A. R.), sight-testing obturator . . . 382
Glioma, hereditary, of the retina (A. Hill Griffith) . . 242
Greeves (R. A.), choroidal degeneration . . . 381
——— mass at the right macula (? tubercular) . . 381
——— nodular elevations on the surface of the iris . . 381
Griffith (A. Hill), hereditary glioma of the retina . . 242
Grimsdale (H.), congenital buphthalmos, with enlargement of the arteries of the retina . . . 175
——— discussion on employment of the blind . . 150
——— essential shrinking of the conjunctiva . . 381

Hepburn (M. L.), papilloma of the conjunctiva . . 190
Hereditary optic atrophy (Leber's disease), (W. B. Inglis Pollock) 247
Herpes zoster, affecting the ciliary nerves (M. S. Mayou) . 226
Hudson (A. C.), neuro-fibromatosis of the eyelids . . 381
——— unilateral melanosis of a segment of the iris . . 174
Hypertension, sclero-corneal trephining for (J. Gray Clegg) . 308
Hypotony, sequelæ of (E. Treacher Collins) . 281

Injuries 176
Intra-ocular tension (E. Treacher Collins) . . . 281
Irido-cyclitis of obscure origin (D. L. Davies) . . 220
Irido-cycloplegia, case of, with uveo-parotitis (George Mackay) 208
Iris, nodular elevations on the surface of the (R. A. Greeves) . 381
——— unilateral melanosis of a segment of the (A. C. Hudson) 174

Jessop (W. H.), death of 1

Lantern slides, regulations for the loan of . . xxxvii
Lawford (J. B.), equatorial staphyloma of an unknown ætiology 381

PAGE

Lawson (Arnold), case of detachment of the retina at the ora serrata, 231; Mr. Treacher Collins' pathological report on 234
——— discussion on advancement operations . . 368
——— ——— employment of the blind . . . 144
——— ——— the value of the ophthalmometer in estimating the refraction of the eye 346
Leber's disease associated with changes in the sella turcica (J. Herbert Fisher) 251
——— two cases of (hereditary optic atrophy) (W. B. Inglis Pollock) 247
——— without family tendency (J. Herbert Fisher) . . 256
Leuco-sarcoma of choroid and epithelioma of lip (J. A. Valentine) 229
Lens, diseases of the 264
Library Rules xxxvi

Mackay (George), case of "uveo-parotitis" with irido-cyclo-plegia 208
——— discussion on concussion injuries of the visual apparatus 128
——— ——— conditions affecting standards of vision in an army 364
McMullen (W. H.), discussion on conditions affecting standards of vision in an army 364
——— evolution of the ophthalmoscope . . . 324
Macula, holes at the, relation of subchoroidal hæmorrhage from concussion to (E. Treacher Collins) . . . 112
——— pigmentary disturbances at the; relation of subchoroidal hæmorrhage from concussion to (E. Treacher Collins) . 112
——— right, mass at the, ? tubercular (R. A. Greeves) . 381
——— symmetrical pigmented changes at the (F. A. C. Tyrrell) 237
Mayou (M. S.), ? colloid nodules in the choroid in a girl . 227
——— discussion on case of opaque nerve-fibres . . 170
——— ——— papilloma of the conjunctiva . . 190
——— orbital suppuration 191
——— pulsating exophthalmos as a result of a wound of the head 188
——— two cases of herpes zoster affecting the ciliary nerves . 226
Melanosis, unilateral, of a segment of the iris (A. C. Hudson) . 174
Members, list of xi
Mooren's ulcer treated with a conjunctival flap (F. A. C. Tyrrell) 205
Museum, list of exhibitors 381

Nephritis, war, fundus lesions in cases of (J. Jameson Evans) . 238

PAGE
Nerve-fibres, opaque, unusual case of (Leslie Buchanan) . 166
Nettleship prize xxxviii
Neuro-fibromatosis of the eyelids (A. C. Hudson) . . . 381

Officers and council ix
Opaque nerve-fibres, unusual case of (Leslie Buchanan) . 166
Operations, advancement, remarks upon (C. Worth) . . 365
Ophthalmometer, value of the, in estimating the refraction of the eye (Ernest Clarke) 343
Ophthalmoscope, evolution of the (W. H. McMullen) . . 324
Optic disc, small tortuous veins on the (E. Treacher Collins) . 171
——— nerve and retina, diseases of the . . . 231
Orbit and eyelid, diseases of the 188
Orbital suppuration (M. S. Mayou) . . . 191
Ora serrata, detachment of the retina at the (A. Lawson) . 231
Ormond (Capt. A. W.), discussion on conditions affecting standards of vision in an army . . . 363
——— ——— employment of the blind . . . 157
——— introduction to a discussion on concussion injuries of the visual apparatus 60

Papilloma of the conjunctiva (M. L. Hepburn) . . 190
Parsons (J. Herbert), aniridia 381
——— apocritic principle and the evolution of visual perceptions 333
——— congenital cataract, heterochromia, and persistent hyaloid artery 381
——— discussion on detachment of the retina at the ora serrata 236
——— ——— the sequelæ of hypotony . . . 308
Paton (Leslie), discussion on treatment of Mooren's ulcer . 206
——— functional spasm of accommodation . . 370
Pearson (Sir Arthur), introduction to discussion on the employment of the blind 133
Pollock (W. B. Inglis), discussion on cases of Leber's disease 262
——— discussion on conditions affecting standards of vision in an army 364
——— two cases of Leber's disease (hereditary optic atrophy) 247
Presidential address (E. Treacher Collins) . . . 1
Presidents of the Society x
Protection of the eyes in warfare (Capt. R. R. Cruise) . 176

PAGE
Refraction of the eye, value of the ophthalmometer in estimating the (Ernest Clarke) 343
Resolution on the conditions affecting the standards of vision in the British Army 364
——— protection of eyes in warfare . . . 184
Retina and optic nerve, diseases of the . . . 231
——— detachment of the, at the ora serrata (A. Lawson) . 231
——— hereditary glioma of the (A. Hill Griffith) . . 242
——— pigmentation of the, relation of subchoroidal hæmorrhage from concussion to (E. Treacher Collins) . . 112
Ridley (N. C.), discussion on concussion injuries of the visual apparatus 129
——— ——— pulsating exophthalmos . . . 187
Rockliffe (W. C.) discussion on employment of the blind . 139
Rowan (John), discussion on employment of the blind . 159
——— ——— the value of the ophthalmometer in estimating the refraction of the eye 347
——— ——— treatment of Mooren's ulcer . . 207
Rules of the Society xxxiv

St. Dunstan's College for the Blind, visit to . . 133
Sclero-corneal trephining for hypertension (J. Gray Clegg) . 308
Sella turcica, Leber's disease associated with the (J. Herbert Fisher) 251
Sight-testing obturator (A. R. Galloway) . . . 382
Smith (Priestley), discussion on hereditary glioma of the retina 246
——— ——— protection of the eyes in warfare . . 183
Suppuration, orbital (M. S. Mayou) . . . 191

Taylor (S. Johnson), discussion on hereditary glioma of the retina 246
——— ——— the sequelæ of hypotony . . . 307
Trephining, sclero-corneal, for hypertension (J. Gray Clegg) . 308
Trustees of the Society x
Tuckett (I. L.), functional blepharospasm in an old man . 188
Tyrrell (F. A. C.), Mooren's ulcer treated with a conjunctival flap 205
——— symmetrical pigmented changes at the macula . 237

Ulcer, Mooren's, treated with a conjunctival flap (F. A. C. Tyrrell) 205
Uveal tract, diseases of the 208

PAGE

"Uveo-parotitis," case of, with irido-cycloplegia (George Mackay) 208

Valentine (J. A.), leuco-sarcoma of choroid and epithelioma of lip in the same patient 229

Visual apparatus, concussion injuries of the; discussion on, introduced by Capt. A. W. Ormond 60

——— perceptions, apocritic principle and the evolution of (J. Herbert Parsons) 333

Vision in an army, conditions affecting standards of (W. Wallace 349

Wallace (Capt. W.), conditions affecting standards of vision in an army 349

——— resolution *re* the above 364

War nephritis, fundus lesions in cases of (J. Jameson Evans) . 238

Warfare, concussion injuries of the visual apparatus in, discussion on, introduced by Capt. A. W. Ormond . . 60

——— protection of the eyes in: resolution on the subject . 184

——— protection of the eyes in (Capt. R. R. Cruise) . 176

Whitehead (A. L.) discussion on concussion injuries of the visual apparatus 126

——— ——— papilloma of the conjunctiva . . 191

——— ——— pulsating exophthalmos . . . 187

Wilson (S. A. Kinnier), discussion on concussion injuries of the visual apparatus in warfare, of central origin . 92

Worth (Claud), remarks upon advancement operations . 365

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